Medical Laboratory Journal
Online ISSN: 2538-4449
Golestan University of Medical Sciences

Original Article

Effects of High-Intensity Interval Training on Adropin, Blood Glucose Markers, Insulin Resistance

Fatemeh Khodadadi
(MSc student) Department of Exercise Physiology, Faculty of Sports Sciences, Ferdowsi University of Mashhad, Mashhad, Iran

Seyed Reza Attarzadeh Hosseini
(PhD) Department of Exercise Physiology, Faculty of Sports Sciences, Ferdowsi University of Mashhad, Mashhad, Iran

Mohammad Mosaferi
(PhD) Department of Exercise Physiology, Faculty of Sports Sciences, Ferdowsi University of Mashhad, Mashhad, Iran

Corresponding author: Seyed Reza Attarzadeh Hosseini

Email: attarzadeh@um.ac.ir

Address: Department of Exercise Physiology, Faculty of Sports Sciences, Ferdowsi University of Mashhad, Mashhad, Iran

Received: 2020/09/21
Revised: 2020/03/23
Accepted: 2020/03/28

DOI: 10.29252/mlj.16.2.27

ABSTRACT

Background and objectives: Previous investigations have shown that physical activity can improve insulin sensitivity and body composition by reducing the concentration of inflammatory biomarkers. The study aimed at evaluating effects of eight weeks of resistance training and high-intensity interval training on adropin, blood glucose markers, and body composition in overweight females.

Methods: sixteen overweight females (mean age: 30 ± 4.3 years and body mass index= 29 ± 2.6 kg/m^2) were randomly assigned to a resistance training group (n=8) and a high-intensity interval training group (n=8). Participants in both groups trained three times a week for eight weeks. Body composition and serum level of blood markers were determined at baseline and after the last training session.

Results: Body mass, body fat percentage, and waist-hip ratio decreased significantly in both groups ($p<0.05$). VO_{2max} significantly increased in both groups, while the changes in the resistance training group were greater than in the high-intensity interval training group ($p<0.05$). Insulin and HOMA-IR concentrations decreased significantly in the resistance training group ($p<0.05$).

Conclusion: Eight weeks of both training procedures could significantly decrease body composition markers. However, the training duration was not sufficient to alter fating blood sugar or adropin concentrations.

Keywords: Body composition, obesity, resistance training, high-intensity interval training.

INTRODUCTION

Obesity and overweight are the major contributors to many diseases such as type 2 diabetes (T2D) and cardiovascular disease by mainly increasing risk of chronic inflammation, insulin resistance, dyslipidemia, and hypertension (1, 2). Various factors such as sedentarism, smoking, and excessive alcohol consumption can increase the severity of the mentioned diseases, especially T2D (3). Insulin resistance, an indicator of T2D, refers to a condition in which physiological concentrations of insulin are less effective in controlling blood sugar (4, 5). In this condition, cells become resistant to insulin, which is tightly linked with development of T2D (6, 7). On the other hand, adipose tissue has been shown to play a key role in insulin resistance through the irregular secretion of numerous inflammatory biomarkers including apelin, tumor necrosis factor, leptin, adiponectin, angiotensin, resistin, etc. (8-13). In addition, adropin has been recently regarded as a metabolic hormone involved in the regulation of lipid metabolism that could be reduced in obese individuals (14-17). Research shows that transgenic overexpression or systemic adropin treatment improves diet-induced obesity, insulin resistance, and glucose tolerance (18). More specifically, adropin attenuates body mass and obesity-associated metabolic disorders (16). Overall, adropin could be considered as a promising target for developing therapies against the metabolic disorders associated with obesity (18).

Previous investigations have shown that physical activity can improve insulin sensitivity and body composition by reducing the concentration of inflammatory biomarkers (19). For instance, endurance training (ET) has been shown to decrease adiposity, which negatively correlates with inflammatory biomarkers (20, 21). Particularly, the intensity of ET has been the subject of interest due to its impact on health-related outcomes of this exercise (22). High-intensity interval training (HIIT) is an effective method to stimulate several metabolic adaptations, such as decreased inflammatory markers in obese individuals, which in turn could increase insulin sensitivity while decreasing insulin resistance (9). In this regard, it has been recently reported that continuous moderate-intensity exercise for 30 minutes does not affect body fat percentage compared to HIIT (9). In one study, eight weeks of ET significantly increased serum adropin concentrations in obese adults (23). Fujie et al. (2015) also reported that eight weeks of ET at 60-70% of maximum heart rate increased adropin concentrations in healthy middle-aged and elderly individuals (24). Furthermore, Alizadeh et al. (2018) demonstrated a significant increase in adropin concentrations following ET in overweight females (25). Given the lack of studies on the effects of resistance training (RT) and HIIT on serum concentrations of adropin in obese or overweight individuals, we aimed to compare effects of RT and HIIT on body composition, insulin, and adropin concentrations in overweight females.

MATERIALS AND METHODS

Twenty-four overweight and obese females (mean age: 30 ± 4.3 years, mean height: 164.8 ± 4.4 cm) participated in the present study. Inclusion criteria included age of between 25 and 35 years and body mass index (BMI) of 25-35. Exclusion criteria were having cardiovascular disease, diabetes, hypertension, sleep disorders, and history of regular exercise in the last six months. Written consent was obtained from all subjects before participation in the study. The study protocol was approved by the Institutional Human Subject Committee of Ferdowsi University of Mashhad and carried out in accordance with the Declaration of Helsinki.

Before baseline measurements, the participants were familiarized with the experiments and procedures. Then, they were randomly assigned into a RT group (n=8) and a HIIT group (n=8). The allocation was stratified by BMI of <25.0 or ≥ 25.0 kg/m^2, and the sequence was randomized by a computer. Measurements were made at baseline and after eight weeks of training at a specific time. The subjects were instructed not to alter their regular lifestyle and dietary habits during the study. Maximal strength testing was carried out 24 hours after the body composition measurement. For this purpose, the subjects first warmed up for 10 minutes and then performed two attempts to lift heaviest weight, and the number of repetitions was recorded. The number of repetitions to fatigue did not exceed 10. There were 3-5-minutes of resting

between each attempt. The participant's maximal strength was predicted using the following formula: one-repetition maximum (1RM) = weight/ (1.0278–0.0278×reps) (26).
Subjects in the RT group completed the supervised training three times a week for eight weeks, with at least 48 hours intervals. Before the training session, the subjects started with 10 minutes of general and specific warm-up activities (slow running, stretching and light RT). The training session included four sets of lower and upper limb exercises with 70% of 1RM with 10–12 repetitions. There were 60-second and 2-minute rest intervals between the sets and exercises, respectively. To verify the principle of overload, the following formula was used to predict the 1RM and to determine the exercise load before the start of the training period, on the fourth week, and at the end of the eighth week (27): 1RM = W/ [1.0278(0.0278.r)] (26).
Subjects in the HIIT group also performed 30 minutes of supervised training three times a week for eight weeks, with at least 48 hours intervals. Each HIIT session began with a 5-minute warm-up, followed by 3 or 4 and 5 to 10 minutes short sprints, mixed conditioning, long sprints, power-short, and power long exercises.
The participants were required to empty bladder within 30 minutes of arriving at the laboratory. Body mass (BM) was measured using a digital scale (Rohs, China) to the nearest 0.1 kg. Height was measured with a stadiometer (Seca, Germany) to the nearest 0.1 cm. Finally, BMI, body fat percentage (BFP), skeletal muscle mass (SMM), and waist hip ratio (WHR) were determined using a multi-frequency bioelectrical impedance device (Inbody 720, South Korea). Fasting blood samples (5 ml) were taken from the cubital vein using standard procedures. Blood sampling was performed 48 hours before the first exercise session and 48 hours after the last exercise session. Serum was separated and stored at –80 °C for later analysis. Serum concentration of adropin was measured using a commercial enzyme-linked immunosorbent assay (ELISA) kit (Eastbiopharm, sensitivity: 2.4 ng/L). Fasting blood sugar (FBS) was measured by glucose oxidase method using a commercial kit (Pars Azmun, sensitivity: 5 mg/dl). Blood insulin concentration was measured using a commercial radioimmunoassay kit (Demeditec, sensitivity: 1.76 micIU/ml). Insulin resistance was also calculated using the following equation:
HOMA-IR = [glucose (nmol/L) * insulin (µU/mL)/22.5](28) Normality of data was confirmed by the Shapiro-Wilk test. Changes in body composition and blood parameters were compared using two-way repeated measures ANOVA [group (RT × HIIT) × time (pre × post)]. One-way ANOVA was used to evaluate changes between pre-intervention and post-intervention values where appropriate. Data analysis was carried out in SPSS Statistics (version 24, IBM, USA). Cohen's *d* effect size (ES) was calculated as post-training mean minus pre-training mean/pooled pre-training standard deviation means (29). In addition, ES values of 0.00-0.19, 0.20-0.49, 0.50-0.79, and ≥ 0.80 were considered trivial, small, moderate, and large, respectively. Statistical significance was set at 0.05.

RESULTS

Compliance with RT and HIIT conditions was 66.6 %. Four participants in both groups dropped due to unwillingness to carry out exercise procedures. The values of variables did not differ significantly at baseline ($p>0.05$). A significant effect of time was observed for BM ($p>0.001$), BFP ($p<0.01$), WHR ($p<0.01$), and VO_{2max} ($p<0.01$), while no time × group interaction was detected for the variables overtime ($p>0.05$). The values of BM, BFP, and WHR decreased significantly in both groups. Interestingly, VO_{2max} significantly increased in both groups and the changes in the RT group was greater than HIIT group. However, SMM, adropin and FBS did not change significantly in the study groups ($p>0.05$). Insulin and HOMA-IR concentrations decreased significantly in the RT group (Table 1).

DISCUSSION

The present study aimed at comparing the effects of eight weeks of RT and HITT on adropin, blood glucose markers, and body composition in overweight females. The beneficial effects of physical activity on body composition indices are well documented (30-32). Traditional ET focuses on longer exercise for longer periods, while HIIT consists of high-intensity, short-term training, and active recovery periods, which has significant advantage over traditional ET in terms of time efficiency (33).

Table 1- Comparison of physiological and biochemical factors between the study groups

Variables	Group	Pre-intervention	Post-intervention	P-value	
				within-groups differences	between groups differences
BM (kg)	HIIT	77.3 ± 7.8	73.5 ± 7.0*	0.001	0.179
	RT	79.4 ± 8.7	77.3 ± 7.8*		
BMI (kg/m^2)	HIIT	28.8 ± 2.3	27 ± 1.7*	0.001	0.210
	RT	29.2 ± 3.1	28.3 ± 3.2*		
BFP (%)	HIIT	38 ± 6.4	36.1 ± 5.9 *	0.003	0.282
	RT	39.3 ± 5	38.3 ± 5.2 *		
SMM (kg)	HIIT	26.2 ± 2.4	25.6 ± 2.1	0.056	0.521
	RT	26.4 ± 2.9	26.1 ± 2.5		
WHR (m)	HIIT	0.89 ± 0.02	0.86 ± 0.01*	0.001	0.989
	RT	0.89 ± 0.05	0.86 ± 0.05*		
VO_{2max} (L.m)	HIIT	1.7 ± 0.3	2.2 ± 0.3*	0.001	0.589
	RT	1.6 ± 0.4	2.4 ± 0.7*		
Adropin (ng/L)	HIIT	51.8 ± 19.4	54.8 ± 11.7	0.470	0.784
	RT	51.2 ± 25.6	57.9 ± 16.2		
FBS (ml/dL)	HIIT	81.5 ± 9.8	75.8 ± 5.8	0.091	0.268
	RT	84.5 ± 8	83.2 ± 9.6		
Insulin (micIU/mL)	HIIT	2.9 ± 1.4	2.6 ± 1.4	0.026	0.099
	RT	3.7 ± 1.2	2.8 ± 1.1*		
HOMA-IR	HIIT	2.9 ± 1.4	2.6 ± 1.4	0.019	0.176
	RT	3.7 ± 1.2	2.8 ± 1.1*		

There is robust evidence that HIIT may pose superior benefits compared to traditional endurance exercise on health markers (34, 35). In our study, HIIT was more effective than RT in decreasing BM and BFP, which seems not surprising. It is hypothesized that elevated excess post-exercise oxygen consumption compared to traditional endurance exercised or RT may account for the decrease in BM and BFP in the HIIT group (36-39).

Furthermore, it seems that post-exercise fat oxidation following HIIT is due to hormonal response (40). Although the hormonal responses were not evaluated in the present study, the lipolytic effects of epinephrine in response to HIIT are well-described (41), which would be expected to stimulate fat loss (42). Another main effect of HIIT is the suppression of appetite (43), which might contribute to the decrease in nutrients intake and the subsequent fat and BM loss. In the present study, SMM did not change significantly following both procedures. It seems that the duration of our RT procedure was not long enough, which could explain the lack of significant effect on SMM after eight weeks. Despite the beneficial effects of HIIT on fat loss, this type of exercise may not be advantageous to RT in augmenting SMM gains due to increased energy expenditure after exercise (44), which increases TSC2 protein may be in results of energy deficiency. The experienced energy deficiency inhibits the mechanistic target of rapamycin complex 1, which is the most important signaling pathway in boosting muscle protein synthesis and increased SMM gains (45).

The higher increase of VO_{2max} in the RT group compared to the HIIT group was an interesting finding of our study. However, research has shown increases in SMM in exercising muscle and blood flow to active musculature as well as cardiac output (SV max and maximal heart rate) and arteriovenous oxygen difference (capillary density and myoglobin concentration) contribute to the RT-induced increase of VO_{2max} (46).

Both RT and HIIT protocols failed to alter adropin or FBS concentrations. However, insulin and HOMA-IR concentrations significantly decreased in the RT group. Adropin regulates nitric oxide synthesis through vascular endothelial growth factor receptor 2. Although research on Adropin is very limited, the elevation of adropin concentrations after aerobic exercise has been demonstrated (23). Adropin is effective in preventing obesity, insulin resistance, and impaired glucose tolerance (18). Thus, it seems that improved body composition through physical activity might affect adopin concentrations. Fujie et al. found that serum adropin concentrations are increased by approximately 75% after eight weeks of regular aerobic exercise (24). Alizadeh et al. insulin resistance but not FBS and adropin concentrations in overweight women (25). In our study, insulin and HOMA-IR decreased

assessed reported that 30 minutes of aerobic exercise could improve insulin levels and significantly in the RT group. This may be due to the appropriate training period and the involvement of large muscle groups in the training protocol that require higher cellular energy compared to small muscle groups (47). The limitations of our study include the small sample size and utilization of bioelectrical impedance to measure body composition, which is not as accurate as dual-energy x-ray absorptiometry (the gold standard technique for body composition measurement); however, previous studies have shown that it is a valid and reliable method (48, 49).

CONCLUSION

Despite application of a proper volume and intensity of both RT and HIIT procedures, only the concentrations of insulin and HOMA-IR changed significantly after eight weeks of RT. However, both exercises similarly affected body composition indices. In addition, RT was not capable of affecting SMM overtime maybe due to short period of study. Conducting future studies with a larger sample size for longer periods could verify our findings.

ACKNOWLEDGMENTS

We are thankful to all participants for their cooperation, especially during the pandemic.

DECLARATIONS

Funding

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

Ethics approvals and consent to participate

Written consent was obtained from all subjects before participation in the study. The study protocol was approved by the Institutional Human Subject Committee of Ferdowsi University of Mashhad and carried out in accordance with the Declaration of Helsinki.

Conflict of interest

The authors declare that there is no conflict of interest regarding publication of this article.

REFERENCES

1. Kannel WB. *Metabolic risk factors for coronary heart disease in women: perspective from the Framingham Study.* American heart journal. 1987;114(2):413-9. [View at Publisher] [DOI:10.1016/0002-8703(87)90511-4] [PubMed] [Google Scholar]

2. Tounian P, Aggoun Y, Dubern B, Varille V, Guy-Grand B, Sidi D, et al. *Presence of increased stiffness of the common carotid artery and endothelial dysfunction in severely obese children: a prospective study*. Lancet. 2001 27;358(9291):1400-4. [View at Publisher] [DOI:10.1016/S0140-6736(01)06525-4] [PubMed] [Google Scholar]

3. Ruth KS, Day FR, Hussain J, Martínez-Marchal A, Aiken CE, Azad A, et al. *Genetic insights into biological mechanisms governing human ovarian ageing*. Nature. 2021 ;596(7872):393-397. [DOI:10.1097/00006254-200203000-00018] [PubMed] [Google Scholar]

4. Di Meo S, Iossa S, Venditti P. *Improvement of obesity-linked skeletal muscle insulin resistance by strength and endurance training.* J Endocrinol. 2017 ;234(3):R159-R181. [DOI:10.1530/JOE-17-0186] [PubMed] [Google Scholar]

5. 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): 625-32. [View at Publisher] [DOI:10.1530/EJE-07-0223] [PubMed] [Google Scholar]

6. Nishida T, Tsuji S, Tsujii M, Arimitsu S, Haruna Y, Imano E, et al. *Oral glucose tolerance test predicts prognosis of patients with liver cirrhosis.* American Journal of Gastroenterology. 2006; 101(1): 70-5. [DOI:10.1111/j.1572-0241.2005.00307.x] [PubMed] [Google Scholar]

7. Judith A. *Insulin resistance/Diabetes and HIV and hepatitis C.11th Annual Retrocirus Conference (CROI-Conference on Retroviruses and pportunistic Infections).* San Francisco. 2004. [View at Publisher]

8. Spiegelman BM, Flier JS. *Adipogenesis and obesity: rounding out the big picture.* Cell. 1996; 87(3): 377-89. [DOI:10.1016/S0092-8674(00)81359-8] [PubMed] [Google Scholar]

9. Friedman JM, Halaas JL. *Leptin and the regulation of body weight in mammals.* Nature. 1998;395(6704):763. [View at Publisher] [DOI:10.1038/27376] [Google Scholar]

10. Hotamisligil GS, Shargill NS, Spiegelman BM. *Adipose expression of tumor necrosis factor-alpha: direct role in obesity-linked insulin resistance*. Science. 1993;259(5091):87-91. [DOI:10.1126/science.7678183] [PubMed] [Google Scholar]

11. Hotamisligil GS, Spiegelman BM. *Tumor necrosis factor α: a key component of the obesity-diabetes link.* Diabetes. 1994;43(11):1271-8. [View at Publisher] [DOI:10.2337/diab.43.11.1271] [PubMed] [Google Scholar]

12. Shimomura I, Funahasm T, Takahashi M, Maeda K, Kotani K, Nakamura T, et al. *Enhanced expression of PAI-1 in visceral fat: Possible contributor to vascular disease in obeisty.* Nature medicine. 1996; 2(7): 800-3. [View at Publisher] [DOI:10.1038/nm0796-800] [PubMed] [Google Scholar]

13. Hotamisligil GS, Arner P, Caro JF, Atkinson RL, Spiegelman BM. *Increased adipose tissue expression of tumor necrosis factor-alpha in human obesity and insulin resistance.* The Journal of clinical investigation. 1995;95(5):2409-15. [DOI:10.1172/JCI117936] [PubMed] [Google Scholar]

14. Schnabel RB, Wild PS, Schulz A, Zeller T, Sinning CR, Wilde S, et al. *Multiple endothelial biomarkers and noninvasive vascular function in the general population:* the Gutenberg Health Study. Hypertension. 2012; 60(2): 288-95. [DOI:10.1161/HYPERTENSIONAHA.112.191874] [PubMed] [Google Scholar]

15. Lovren F, Pan Y, Quan A, Singh KK, Shukla PC, Gupta M, et al. *Adropin is a novel regulator of endothelial function*. Circulation. 2010;122(11_suppl_1):S185-S92. [View at Publisher] [DOI:10.1161/CIRCULATIONAHA.109.931782] [PubMed]

16. Lovren F, Pan Y, Quan A, Singh KK, Shukla PC, Al-Omran M, et al. *Adropin is a Novel Regulator of Endothelial Function Through the VEGFR2-PI3K-Akt and VEGFR2-Erk1/2 Pathways. Am Heart Assoc*; 2010; 122(21): [DOI:10.1161/CIRCULATIONAHA.109.931782] [Google Scholar]

17. Butler AA, Tam CS, Stanhope KL, Wolfe BM, Ali MR, O'Keeffe M, et al. *Low circulating adropin concentrations with obesity and aging correlate with risk factors for metabolic disease and increase after gastric bypass surgery in humans*. The Journal of Clinical Endocrinology & Metabolism. 2012; 97(10): 3783-91. [DOI:10.1210/jc.2012-2194] [PubMed] [Google Scholar]

18. Kumar KG, Trevaskis JL, Lam DD, Sutton GM, Koza RA, Chouljenko VN, et al. *Identification of adropin as a secreted factor linking dietary macronutrient intake with energy homeostasis and lipid metabolism*. Cell metabolism. 2008; 8(6): 468-81. [View at Publisher] [DOI:10.1016/j.cmet.2008.10.011] [PubMed] [Google Scholar]

19. Frøsig C. *Effect of acute exercise and exercise training on insulin stimulated glucose uptake in human skeletal muscle*. 2012.

20. Elmer DJ, Laird RH, Barberio MD, Pascoe DD. *Inflammatory, lipid, and body composition responses to interval training or moderate aerobic training*. European journal of applied physiology. 2016;116(3):601-9. [View at Publisher] [DOI:10.1007/s00421-015-3308-4] [PubMed] [Google Scholar]

21. Devries MC, Hamadeh MJ, Glover AW, Raha S, Samjoo IA, Tarnopolsky MA. *Endurance training without weight loss lowers systemic, but not muscle, oxidative stress with no effect on inflammation in lean and obese women*. Free radical biology and medicine. 2008;45(4):503-11. [View at Publisher] [DOI:10.1016/j.freeradbiomed.2008.04.039] [PubMed] [Google Scholar]

22. Poirier P, Després J-P. *Exercise in weight management of obesity*. Cardiology clinics. 2001; 19(3): 459-70. [DOI:10.1016/S0733-8651(05)70229-0] [PubMed]

23. Fujie S, Hasegawa N, Kurihara T, Sanada K, Hamaoka T, Iemitsu M. *Association between aerobic exercise training effects of serum adropin level, arterial stiffness, and adiposity in obese elderly adults*. Applied Physiology, Nutrition, and Metabolism. 2017;42(1):8-14. [View at Publisher] [DOI:10.1139/apnm-2016-0310] [PubMed] [Google Scholar]

24. Fujie S, Hasegawa N, Sato K, Fujita S, Sanada K, Hamaoka T, et al. *Aerobic exercise training-induced changes in serum adropin level are associated with reduced arterial stiffness in middle-aged and older adults.* American Journal of Physiology-Heart and Circulatory Physiology. 2015;309(10):H1642-H7. [View at Publisher] [DOI:10.1152/ajpheart.00338.2015] [PubMed] [Google Scholar]

25. Alizadeh R, Golestani N, Moradi L, Rezaeinejad N. *Effect of aerobic exercise with maximal fat oxidation intensity, on adropin and insulin resistance among overweight women.* Iranian Journal of Endocrinology and Metabolism. 2018;20(2):81-8. [View at Publisher] [Google Scholar]

26. Murach KA, Bagley JR. *Skeletal muscle hypertrophy with concurrent exercise training: contrary evidence for an interference effect.* Sports medicine. 2016;46(8):1029-39. [View at Publisher] [DOI:10.1007/s40279-016-0496-y] [PubMed] [Google Scholar]

27. Bagheri R, Rashidlamir A, Motevalli MS, Elliott BT, Mehrabani J, Wong A. *Effects of upper-body, lower-body, or combined resistance training on the ratio of follistatin and myostatin in middle-aged men*. European journal of applied physiology. 2019;119(9):1921-31. [View at Publisher] [DOI:10.1007/s00421-019-04180-z] [PubMed] [Google Scholar]

28. Matthews D, Hosker J, Rudenski A, Naylor B, Treacher D, Turner R. *Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man*. Diabetologia. 1985;28(7):412-9. [View at Publisher] [DOI:10.1007/BF00280883]

29. Cohen J. *A power primer*. Psychological bulletin. 1992;112(1):155. [View at Publisher] [DOI:10.1037/0033-2909.112.1.155] [Google Scholar]

30. Shaw KA, Gennat HC, O'Rourke P, Del Mar C. *Exercise for overweight or obesity.* Cochrane database of systematic reviews. 2006. [DOI:10.1002/14651858.CD003817.pub3] [Google Scholar]

31. Ohkawara K, Tanaka S, Miyachi M, Ishikawa-Takata K, Tabata I. *A dose-response relation between aerobic exercise and visceral fat reduction: systematic review of clinical trials.* International journal of obesity. 2007; 31(12): 1786-97. [View at Publisher] [DOI:10.1038/sj.ijo.0803683] [PubMed] [Google Scholar]

32. Verheggen R, Maessen M, Green DJ, Hermus A, Hopman M, Thijssen D. *A systematic review and meta-analysis on the effects of exercise training versus hypocaloric diet: distinct effects on body weight and visceral adipose tissue.* Obesity Reviews. 2016;17(8):664-90. [View at Publisher] [DOI:10.1111/obr.12406] [PubMed] [Google Scholar]

33. Trost SG, Owen N, Bauman AE, Sallis JF, Brown W. *Correlates of adults' participation in physical activity: review and update.* Medicine & science in sports & exercise. 2002;34(12):1996-2001. [View at Publisher] [DOI:10.1097/00005768-200212000-00020] [PubMed] [Google Scholar]

34. Bacon AP, Carter RE, Ogle EA, Joyner MJ. *VO 2 max trainability and high intensity interval training in humans: a meta-analysis.* PloS one. 2013;8(9):e73182. [View at Publisher] [DOI:10.1371/journal.pone.0073182] [PubMed] [Google Scholar]

35. Milanović Z, Sporiš G, Weston M. *Effectiveness of high-intensity interval training (HIT) and continuous endurance training for VO 2max improvements: a systematic review and meta-analysis of controlled trials.* Sports medicine. 2015; 45(10): 1469-81. [View at Publisher] [DOI:10.1007/s40279-015-0365-0] [PubMed] [Google Scholar]

36. Chan HH, Burns SF. *Oxygen consumption, substrate oxidation, and blood pressure following sprint interval exercise.* Applied Physiology, Nutrition, and Metabolism. 2013; 38(2): 182-7. [View at Publisher] [DOI:10.1139/apnm-2012-0136] [PubMed] [Google Scholar]

37. Gore C, Withers R. *The effect of exercise intensity and duration on the oxygen deficit and excess post-exercise oxygen consumption.* European journal of applied physiology and occupational physiology. 1990;60(3):169-74. [View at Publisher] [DOI:10.1007/BF00839153] [PubMed] [Google Scholar]

38. Hazell TJ, Olver TD, Hamilton CD, Lemon PW. *Two minutes of sprint-interval exercise elicits 24-hr oxygen consumption similar to that of 30 min of continuous endurance exercise.* International journal of sport nutrition and exercise metabolism. 2012; 22(4): 276-83. [DOI:10.1123/ijsnem.22.4.276] [PubMed] [Google Scholar]

39. Laforgia J, Withers RT, Gore CJ. *Effects of exercise intensity and duration on the excess post-exercise oxygen consumption.* Journal of sports sciences. 2006; 24(12): 1247-64. [View at Publisher] [DOI:10.1080/02640410600552064] [PubMed] [Google Scholar]

40. Boutcher SH. *High-intensity intermittent exercise and fat loss.* Journal of obesity. 2011;2011. [View at Publisher] [DOI:10.1155/2011/868305] [PubMed] [Google Scholar]

41. Zouhal H, Jacob C, Delamarche P, Gratas-Delamarche A. *Catecholamines and the effects of exercise, training and gender.* Sports medicine. 2008;38(5):401-23. [View at Publisher] [DOI:10.2165/00007256-200838050-00004] [Google Scholar]

42. Galster AD, Clutter WE, Cryer PE, Collins JA, Bier D. *Epinephrine plasma thresholds for lipolytic effects in man: measurements of fatty acid transport with [l-13C] palmitic acid.* The Journal of clinical investigation. 1981;67(6):1729-38. [View at Publisher] [DOI:10.1172/JCI110211] [PubMed] [Google Scholar]

43. Williams CB, Zelt JG, Castellani LN, Little JP, Jung ME, Wright DC, et al. *Changes in mechanisms proposed to mediate fat loss following an acute bout of high-intensity interval and endurance exercise*. Applied Physiology, Nutrition, and Metabolism. 2013; 38(12): 1236-44. [View at Publisher] [DOI:10.1139/apnm-2013-0101] [PubMed] [Google Scholar]

44. Paoli A, Moro T, Marcolin G, Neri M, Bianco A, Palma A, et al. *High-Intensity Interval Resistance Training (HIRT) influences resting energy expenditure and respiratory ratio in non-dieting individuals*. Journal of translational medicine. 2012;10(1):237. [View at Publisher] [DOI:10.1186/1479-5876-10-237] [PubMed] [Google Scholar]

45. Laplante M, Sabatini DM. *mTOR signaling at a glance.* Journal of cell science. 2009; 122(20): 3589-94. [View at Publisher] [DOI:10.1242/jcs.051011] [PubMed] [Google Scholar]

46. Ozaki H, Loenneke JP, Thiebaud RS, Abe T. *Resistance training induced increase in VO 2 max in young and older subjects*. European Review of Aging and Physical Activity. 2013; 10(2): 107-16. [View at Publisher] [DOI:10.1007/s11556-013-0120-1] [Google Scholar]

47. Reis VM, Garrido ND, Vianna J, Sousa AC, Alves JV, Marques MC. *Energy cost of isolated resistance exercises across low-to high-intensities.* PloS one. 2017;12(7):e0181311. [View at Publisher] [DOI:10.1371/journal.pone.0181311] [PubMed] [Google Scholar]

48. Ling CH, de Craen AJ, Slagboom PE, Gunn DA, Stokkel MP, Westendorp RG, et al. *Accuracy of direct segmental multi-frequency bioimpedance analysis in the assessment of total body and segmental body composition in middle-aged adult population.* Clinical nutrition. 2011;30(5):610-5. [View at Publisher] [DOI:10.1016/j.clnu.2011.04.001] [PubMed] [Google Scholar]

49. Jackson A, Pollock ML, Graves JE, Mahar M. *Reliability and validity of bioelectrical impedance in determining body composition*. Journal of Applied Physiology. 1988;64(2):529-34. [View at Publisher] [DOI:10.1152/jappl.1988.64.2.529] [PubMed] [Google Scholar]

How to Cite:
Khodadadi F, Attarzadeh Hosseini SR, Mosaferi M [Effects of High-Intensity Interval Training on Adropin, Blood Glucose Markers, Insulin Resistance]. mljgoums. 2022; 16(2): 27-33 DOI: 10.29252/mlj.16.2.27