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The Effect of Resistance Training On Serum Adropin, Fetuin-A, and Insulin Resistance in Overweight Women

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ABSTRACT

Introduction: Overweight is associated with disturbances in serum levels of adipokines and cytokines that affect metabolism and insulin action. In this study, we aimed to assess the effect of resistance training on serum adropin, fetuin-A, and insulin resistance in sedentary overweight women.

Methods: In this quasi-experimental study, 24 overweight women aged 25-35 (BMI between 26 and 30) were randomly divided into exercise and control groups. The exercise group participated in an 8-week resistance training program (three times per week), while the control group continued their sedentary lifestyle. Fasting blood samples were obtained from the brachial vein before the training program and 48 hours after the final exercise session to measure serum adropin, fetuin-A, glucose, insulin, and insulin resistance. Results were then compared between the two groups.

Results: Resistance training resulted in a significant decrease in serum fetuin-A (p=0.014), glucose (p=0.003), and insulin resistance (p=0.011), and a significant increase in serum adropin (p=0.021). No significant changes were observed in the control group for fetuin-A (p=0.413), glucose (p=0.351), adropin (p=0.539), or insulin resistance (p=0.137).

Conclusion: Based on our findings, improvements in insulin resistance can be attributed to changes in serum adropin and fetuin-A in response to resistance training. Identifying the mechanisms responsible for these changes requires further studies in this field.

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Introduction

The prevalence of obesity and overweight has become an epidemic and is a strong independent predictor of mortality. The increase in morbidity and mortality observed in individuals with obesity and overweight has been primarily attributed to the rise in the incidence of type 2 diabetes (T2D) and cardiovascular diseases (CVD) (1). The indiscriminate consumption of energy, sedentary lifestyle, and aging are key contributors to the growing prevalence of obesity, overweight, sarcopenia, metabolic syndrome, type 2 diabetes, and cardiovascular diseases (2). Adipokines, active molecules secreted by adipose tissue, play a crucial role in regulating appetite and satiety, inflammation, energy expenditure, insulin resistance, insulin secretion, glucose and lipid metabolism, and atherosclerosis (3). Evidence suggests that skeletal muscles and the liver also function as endocrine organs by secreting myokines and hepatokines. New research into these organokines (adipokines, myokines, and hepatokines) may lead to the development of novel biomarkers and therapeutic strategies for cardiometabolic diseases (4).

Among them, Fetuin-A is a glycoprotein primarily synthesized by liver cells and released into the bloodstream. Fetuin-A consists of a long A chain (282 amino acids) and a short B chain (27 amino acids), connected by a short linker peptide composed of 40 amino acids. This protein, named initially "fetuin," was later renamed fetuin-A. It is a phosphorylated hepatic glycoprotein that circulates systemically through the bloodstream, with a serum concentration ranging from 0.5 to 1 gram per liter (5). Fetuin-A is the first known hepatokine that targets the liver and has been identified as a natural inhibitor of the insulin receptor, blocking insulin receptor activation by

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insulin tyrosine kinase and contributing to insulin resistance in rodents. Fetuin-A has also been identified as an endogenous ligand for the TLR4 receptor, where it promotes pro-inflammatory signaling pathways and insulin resistance upon interaction with saturated fatty acids. Fetuin-A levels are elevated in the presence of obesity, metabolic syndrome, and type 2 diabetes and are associated with hepatic steatosis in humans. Increased fetuin-A levels have been introduced as a predictive risk factor for type 2 diabetes and myocardial infarction (4). Adropin is a 76-amino acid peptide hormone. Its role in nutrition and the regulation of its levels is undeniable.

It is primarily secreted by the liver, critical in regulating carbohydrate homeostasis and glucose metabolism (6). Adropin is an essential regulatory component in preventing cardiovascular damage and controls cardiac metabolism and vascular function (6). Studies in animal models have shown that the expression of the adropin gene is reduced under conditions of a high-fat diet and leptin deficiency, which are linked to overweight. Moreover, its overexpression or treatment with adropin leads to glucose intolerance and improvements in insulin resistance. Due to its significant role in carbohydrate and fat metabolism, adropin is key in regulating energy homeostasis and has been identified as a regulator of glucose homeostasis independent of changes in body weight and caloric intake (6). It is hypothesized that adropin reduces insulin resistance and hyperglycemia by enhancing glucose metabolism and increasing insulin sensitivity in target tissues (7).

In summary, the increase in fetuin-A and the decrease in adropin, either independently or in interaction with each other, are associated with increased insulin resistance, or in other words, reduced insulin function and hyperglycemia in obese or overweight individuals. Based on this evidence, it appears that therapeutic interventions aimed at lowering fetuin-A and increasing adropin may improve insulin function and lower blood glucose levels in obese or overweight populations, both healthy and diseased. In a study by Moradi et al. (2021), both six weeks of aerobic exercise and six weeks of high-intensity interval training were found to increase adropin levels and decrease glucose and insulin resistance in overweight men, with a significant correlation observed between changes in adropin and both glucose and insulin resistance (8). Zhang et al. (2017) reported that 12 weeks of aerobic exercise, independent of weight loss, significantly enhanced adropin levels and endothelial function in obese adult men (9). In another study by Kermani and colleagues (2021), eight weeks of resistance training increased adropin and reduced glucose and insulin resistance among overweight men (10). However, in research conducted by Saeidi et al. (2021), while eight weeks of high-intensity interval training significantly reduced fetuin-A in obese women, it did not alter the insulin resistance index (11). Nonetheless, Schultes et al. (2010) and Yang et al. (2011) reported no change in serum fetuin-A (12, 13) in response to various exercise training, while Blumenthal et al. (2017) noted an increase in fetuin-A (14). Despite this evidence, few studies have reported the effect of resistance training on fetuin-A and adropin, especially in overweight women. Therefore, the present study was conducted to determine the impact of resistance training on serum adropin and fetuin-A levels in overweight women.

Materials and Methods

Subjects

The present study used a semi-experimental design with a pre-test and post-test structure. The statistical population comprised overweight women (mean age: 31 ± 5.33 years). A total of 24 overweight women (BMI between 26 and 30) were randomly assigned to either the control group (n=12) or the exercise group (n=12). The exercise group participated in an 8-week resistance training program with three sessions per week, while the control group did not engage in any resistance training.

Inclusion and Exclusion Criteria

Overweight (BMI between 26 and 30) was the main criterion for inclusion in the study. The participants were non-athletes, non-smokers, and non-pregnant and had not engaged in any regular training program during the past 6 months. Additionally, they had not followed any defined diet in the last 6 months, and their weight fluctuation did not exceed one kilogram. Participants with a history of kidney diseases, cancer, or seizures were excluded from the study. Regular participation in training sessions and the absence of any disease that could affect the dependent variables were also inclusion criteria.

Anthropometric Measurements

Anthropometric indicators were measured in both groups before and after the exercise intervention. Height was measured using a wall-mounted stadiometer, without shoes, to the nearest 0.1 cm. Hip and abdominal circumferences were measured at the widest point after a normal exhalation using an inelastic tape measure with an error margin of less than 0.1 cm. Weight was measured using a Seca scale, with an accuracy of 0.5 kg. Body mass index (BMI) was calculated by dividing weight (in kilograms) by height (in square meters). Body fat percentage was assessed using a body composition analyzer (OMRON, Finland).

Resistance Training and Blood Sampling

After the anthropometric measurements, the participants were instructed to visit the laboratory for blood sampling after an overnight fast of 10 to 12 hours between 8 and 9 a.m. They were asked to refrain from intense physical activity for 48 hours before blood sampling. Blood samples (5 ml) were collected from the vein of the left arm of each participant while they were sitting and resting. After serum separation, the samples were stored at -80°C until the variables were measured (pre-test). Resistance training was performed over 8 weeks, with three weekly sessions (15). Before starting resistance exercises, each participant's one-repetition maximum (1RM) was evaluated using the Bareziki formula (16). The modified resistance training protocol was followed, with the participants exercising at an intensity of 55-70% of their 1RM, performing three sets with 5-20 repetitions per set, as outlined in Table 1.

Table1. Distribution of training intensity as a percentage of one repetition maximum during resistance training

Weeks	Set	Repetition	Intensity (1RM)
1	3	15-20	55
2	3	15-20	55
3	3	12-15	60
4	3	12-15	60
5	3	8-12	65
6	3	8-12	65
7	3	5-8	70
8	3	5-8	70

Finally, 48 hours after the last training session, blood samples were collected again under the same conditions as the pre-test (post-test). Fasting glucose was measured using the glucose oxidase method, serum insulin was measured by the ELISA method, and adropin (Sunlang, Korea) and fetuin-A (Eastbiopharm, China) were measured using the ELISA method. Insulin resistance was also calculated using the fasting glucose and insulin values according to the following formula (17).

$$Homa - R = \frac{Fasting\ Insulin\ (\mu U/ml) \times Fasting\ Glucose(mmol/l)}{22.5}$$

Data Analysis

Statistical analyses were performed using the SPSS software package (Version 22.0, SPSS Inc., Chicago, IL, USA). The Kolmogorov-Smirnov test was applied to assess the normality of the data. The independent samples t-test was used to compare variables between groups at baseline. The paired t-test was used to determine intra-group changes in variables. The level of statistical significance was set at $p \leq 0.05$.

Results

Table 2 presents the baseline data for anthropometric measurements and biochemical markers. There were no significant differences in anthropometric indices between the groups at baseline ($P > 0.05$). Similarly, baseline comparisons of glucose ($P = 0.596$), insulin resistance ($P = 0.356$), serum adropin ($P = 0.469$), and serum fetuin-A ($P = 0.751$) showed no significant differences between the groups (Table 2).

To assess the extent of variation in each anthropometric index, the first step involved calculating each variable's change (delta) by comparing pre-test and post-test values within each group. Subsequently, these deltas were compared between the two groups using an independent t-test. The statistical analysis revealed a significant difference in the changes of the variables between the groups ($p < 0.05$), indicating a significant reduction in anthropometric measures such as weight, abdominal circumference, body mass index, and body fat percentage following resistance training in the experimental group. Additionally, within-group comparisons using a paired t-test indicated that all measured anthropometric indices significantly decreased after resistance training compared to baseline levels ($p < 0.05$), as shown in Table 3.

Table 2. Clinical and anthropometrical characteristics of the subjects at baseline.

Variables	Exercise group	Control group	P-value
Age (year)	31.7 ± 5.51	32.2 ± 5.83	*0.457*
Height (cm)	164 ± 5.12	165 ± 1.23	*0.363*
Weight (kg)	77.5 ± 6.14	79.5 ± 1.28	*0.414*
Abdominal circumference (cm)	88 ± 5.24	89 ± 3.97	*0.452*
Body mass index (kg/m2)	28.80 ± 2.11	29.2 ± 2.35	*0.426*
Body fat (%)	33.28 ± 3.10	34.03 ± 3.36	*0.544*
Glucose (mg/dL)	116 ± 15	114 ± 10	*0.596*
Insulin resistance (HOMA-IR)	2.75 ± 0.41	2.62 ± 0.59	*0.356*
Serum Adropin (pg/mL)	4.07 ± 0.18	4.27 ± 0.46	*0.469*
Serum fetuin-A (ng/mL)	1279 ± 54	1306 ± 61	*0.751*

Data represented by independent sample t test.

Table 3. Pre and post-training of anthropometrical indexes of 2 groups (Mean ± SD)

Variables	Exercise group			Control group		
	Pre-training	Post-training	Sig	Pre-training	Post-training	Sig
Weight (kg)	77.5 ± 6.14	79 ± 6.21	*0.021*	79.5 ± 1.28	80 ± 6.28	*0.321*
AC (cm)	88 ± 5.24	86 ± 5.19	*0.19*	89 ± 3.97	90 ± 4.23	*0.416*
BMI (kg/m2)	28.80 ± 2.11	29.37 ± 3.28	*0.011*	29.2 ± 2.35	29.38 ± 2.88	*0.213*
Body fat (%)	33.28 ± 3.10	29.59 ± 3.44	*0.007*	34.03 ± 3.36	34.59 ± 2.58	*0.423*

***AC**: abdominal circumference; **BMI:** body mass index*

Table 4. Pre and post-training of clinical markers of 2 groups

Variables	Exercise group			Control group		
	Pre-training	Post-training	Sig	Pre-training	Post-training	Sig
Glucose (mg/dL)	**116** *(± 15)*	**102** *(± 12)*	*0.003*	**114** *(± 10)*	**117** *(± 12)*	*0.351*
Insulin resistance (HOMA-IR)	**2.75** *(± 0.41)*	**1.99** *(± 0.62)*	*0.011*	**2.62** *(± 0.59)*	**2.80** *(± 0.57)*	*0.137*
Serum Adropin (pg/mL)	**4.07** *(± 0.18)*	**4.96** *(± 0.22)*	*0.021*	**4.27** *(± 0.46)*	**4.26** *(± 0.47)*	*0.539*
Serum fetuin-A (ng/mL)	**1279** (± 54)	**1088** (± 58)	*0.014*	**1306** (± 61)	**1044** (± 63)	*0.413*

**Data represented by paired sample t test.*

To assess the variation in each variable, the difference between pre-test and post-test measurements (delta) was calculated for each group. These deltas were then compared between the groups using an independent t-test. The independent t-tests revealed a statistically significant difference between the groups in the deltas for glucose (p = 0.012) and insulin resistance (p = 0.019). Furthermore, analysis of within-group changes showed that resistance training led to a significant reduction in glucose levels compared to baseline in the exercise group (p = 0.003). In contrast, no significant change was observed in the control group (p = 0.351). Similarly, resistance training significantly reduced insulin resistance from baseline levels in the exercise group (p = 0.011), with no significant change detected in the control group (p = 0.137). In addition, the delta comparison showed significant differences between the groups regarding serum adropin (p = 0.016) and fetuin (p = 0.023). However, the intra-group analysis demonstrated that resistance training significantly elevated a drop in levels compared to baseline in the exercise group (p = 0.021). Similarly, resistance training significantly reduced fetuin levels from baseline in the exercise group (p = 0.014). No changes were observed in the control group of these variables.

Discussion

Increased Fetuin-A and the increase in adropin in response to resistance training are the main findings of the present study. In other words, 8 weeks of resistance training, with three sessions per week, led to a significant increase in adropin and a significant decrease in Fetuin-A in overweight women who previously had a sedentary lifestyle. Another key finding is a reduction in fasting glucose and insulin resistance following resistance training, compared to the group that did not participate in training. These findings are consistent with other studies that have also supported the effectiveness of exercise training on glucose and insulin resistance. Soori et al. (2017) reported a significant reduction in glucose after 12 weeks of resistance training (18). In Jorge et al.'s study (2011), a significant decrease in glucose and improved lipid profile indices were observed after 12 weeks of combined training (19). In Wei

et al.'s study (2013), improvements in glucose metabolism and a reduction in insulin resistance following 10 weeks of swimming training were reported (20). Glans et al. (2009) also reported more significant improvements in blood glucose levels in patients who were more active in resistance exercises (21).

The improvement in glucose metabolism and insulin resistance in response to resistance training in the present study, although likely rooted in several metabolic, enzymatic, and hormonal changes, may be attributed to the roles of Fetuin-A and adropin in insulin function in target tissues such as adipose tissue and skeletal muscles. Previous studies have indicated that this improvement could be due to the reduction in Fetuin-A and the increase in adropin in response to this training method. In this context, it has been suggested that Fetuin-A exacerbates insulin resistance by inhibiting insulin-dependent GLUT4. On the other hand, Fetuin-A may disrupt insulin signaling pathways and inhibit insulin binding to its receptors through downstream mechanisms (22). In Salama et al.'s (2017) study, although no change in fasting glucose and serum Fetuin-A was reported in response to resistance swimming exercise, these researchers observed a decrease in fasting glucose in response to moderate-intensity aerobic exercise in diabetic individuals (23). Researchers have also reported a direct relationship between changes in Fetuin-A and insulin resistance following interventions to reduce body fat mass (24).

Researchers believe that metabolic changes and body composition alterations resulting from weight loss interventions and caloric restriction effectively reduce serum Fetuin-A levels (25,26). Based on this evidence, the decrease in serum Fetuin-A observed in the present study may be attributed to improvements in body composition, particularly the reduction in body fat percentage following resistance training. On the other hand, it appears that in reducing body fat mass, an active organ secreting inflammatory cytokines plays a role in lowering serum Fetuin-A levels. For example, a direct relationship between TNF-α secreted from adipose tissue and serum Fetuin-A has been observed (27). Therefore, it is plausible that the reduction in body fat percentage or mass in response to resistance training by decreasing inflammatory cytokines, such as TNF-α, has contributed to the reduction in Fetuin-A serum levels. Laboratory studies have also highlighted that Fetuin-A increases the expression of inflammatory adipokines such as TNF-α (28), inhibits PPARγ in adipose tissue, and decreases the anti-inflammatory cytokine adiponectin (29), all of which lead to increased insulin resistance, particularly in obese individuals.

Apart from Fetuin-A, resistance training in the present study led to increased serum adropin levels compared to the control group. Clinical studies have shown that decreased expression or lower serum levels of adropin are associated with impaired insulin function and increased insulin resistance. Therefore, growing adropin levels, especially in the presence of obesity, can effectively improve insulin resistance and dyslipidemia. For instance, removing adropin in laboratory mice has led to increased adipose tissue, fasting triglycerides, and insulin resistance (30). Based on this evidence, the decrease in glucose levels and insulin resistance in response to resistance training in the present study can likely be attributed to increased serum adropin induced by the exercise intervention. In this context, clinical studies have identified decreased serum adropin levels as contributing to insulin resistance and other components of metabolic syndrome, such as dyslipidemia (31). In confirmation of our findings, Ramzan Khani et al. (2019) reported an increase in adropin and a significant decrease in insulin resistance, glucose levels, and body fat percentage in response to long-term aerobic exercise in sedentary obese women (32). These researchers attributed the glucose and insulin resistance improvement to increased serum adropin, citing a significant correlation between changes in adropin levels and glucose and insulin resistance following aerobic exercise. Laboratory studies have also highlighted the effective role of a drop-in in improving blood glucose through its effects on enzymes and intermediate metabolites involved in carbohydrates and fat metabolisms, such as pyruvate dehydrogenase (PDH) and carnitine palmitoyltransferase (CPT). On the one hand, a drop in PDH activity enhances the final stages of the glycolysis cycle. On the other hand, inhibiting CPT reduces the transfer of fatty acids to the mitochondria, leading to a decrease in the rate of lipid β-oxidation (33). Despite the evidence mentioned above, although the measurement of adropin and fetuin-A in response to resistance

training is a strength of the present study, drawing a general conclusion in this field requires the assessment of other components that influence insulin function, such as adiponectin, insulin levels, and other factors.

Conclusion

Resistance training improves glucose metabolism and insulin resistance in sedentary, overweight women. Based on the findings of this study and considering the critical role of Fetuin-A and adropin in fat-carbohydrate metabolism, the observed improvements in blood glucose and insulin resistance following resistance training are likely due to a reduction in Fetuin-A and an increase in serum adropin. However, a limitation of the present study is the lack of measurement of other key components involved in insulin action, which is necessary to draw a more comprehensive conclusion in this field.

Declarations

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Conflict of Interest

No conflict of interest has been declared.

Ethical Considerations

This study was approved by the Ethics Committee of Islamic Azad University, South Tehran Branch, Tehran, Iran (Code: IR.IAU.SARI.REC.1403.272).

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Authors' Contributions

All authors have contributed to the entire content of this manuscript and are accountable for all aspects of the work. They have ensured that any questions regarding the accuracy or integrity of the work have been appropriately investigated and resolved, and they have approved the final version for publication.

References

1. Kinlen D, Cody D, O'Shea D. Complications of obesity. QJM: An International Journal of Medicine. 2018; 111(7), 437-43.
2. Mukhopadhyay S, Bhattacharya S. Plasma fetuin-A triggers inflammatory changes in macrophages and adipocytes by acting as an adaptor protein between NEFA and TLR-4. Diabetologia. 2016; 59(4):859-60.
3. Rupérez FJ, Martos-Moreno GÁ, Chamoso-Sánchez D, Barbas C, Argente J. Insulin Resistance in Obese Children: What Can Metabolomics and Adipokine Modelling Contribute? Nutrients. 2020; 12(11):3310.
4. Choi KM. The impact of organokines on insulin resistance, inflammation, and atherosclerosis. Endocrinology and Metabolism. 2016; 31(1):1-6.
5. Gunduz FO, Yildirmak ST, Temizel M, Faki Y, Cakmak M, Durmuscan M, Sezgin F. Serum visfatin and fetuin-a levels and glycemic control in patients with obese type 2 diabetes mellitus. Diabetes Metab J. 2011; 35(5):523-8.
6. Marczuk N, Cecerska-Heryć E, Jesionowska A, Dołęgowska B. Adropin - physiological and pathophysiological role. Postepy Hig Med Dosw (Online). 2016; 70(0):981-8.
7. Braith RW, Beck DT. Resistance exercise: training adaptations and developing a safe exercise prescription. Heart Fail Rev. 2008; 13(1):69-79.
8. Moradi G, Ghahramani M. The effect of aerobic and interval training on adropine levels and insulin resistance in overweight men. Journal of Sport in Biomotor Sciences. 2022; 27(1): 13-23.
9. Zhang H, Jiang L, Yang YJ, Ge RK, Zhou M, Hu H, et al. Aerobic exercise improves endothelial function and serum adropin levels in obese adolescents independent of body weight loss. Sci Rep J. 2017; 7(1):1-8.
10. Kermani S, Alizadeh R, Moradi L. The effect of eight weeks of resistance training on adropin plasma level and insulin resistance index in overweight men. Sport and Exercise Physiology. 2021; 14(1):31-7.
11. Saeedi S, Rezaeian N, Karimi M. Changes in serum levels of fetuin-A and insulin resistance following high intensity interval training in young obese women. Journal of Practical Studies of Biosciences in Sport. 2021; 9(18), 64-76.
12. Schultes B, Frick J, Ernst B, Stefan N, Fritsche A. The effect of 6-weeks of aerobic exercise training on serum fetuin-A levels in non-diabetic obese women. Exp Clin Endocrinol Diabetes. 2010; 118(10):754-6.
13. Yang SJ, Hong HC, Choi HY, Yoo HJ, Cho GJ, Hwang TG, e al. Effects of a three-month combined exercise programme on fibroblast growth factor 21 and fetuin-A levels and arterial stiffness in obese women. Clin Endocrinol (Oxf). 2011 Oct; 75(4):464-9.
14. Blumenthal JB, Gitterman A, Ryan AS, Prior SJ. Effects of Exercise Training and Weight Loss on Plasma Fetuin-A Levels and Insulin Sensitivity in Overweight Older Men. J Diabetes Res. 2017; 2017:1492581.
15. Matheus Amarante do Nascimento, Edilson Serpeloni Cyrino, Fábio Yuzo Nakamura, Marcelo Romanzini, Humberto José Cardoso Pianca, Marcos Roberto Queiróga. Validation of the Brzycki equation for the estimation of 1-RM in the bench press. Rev Bras Med Esporte. 2007; 13(1): 40-2.

16. Ebrahim K, Ghazalian F, Rad AA. The effect of eight weeks combined exercise training on the levels of adropin and VEGFR-2 in obese men with hypertension. Razi Journal of Medical Sciences. 2019;26(8):78-88.
17. McAuley KA, Williams SM, Mann JI, Walker RJ, Lewis-Barned NJ, Temple LA, Duncan AW. Diagnosing insulin resistance in the general population. Diabetes Care. 2001; 24(3):460-4.
18. Soori R, Rashidi M, Choobineh S, Ravasi A A, Baesi K, Rashidy-Pour A. Effects of 12 weeks resistant training on MTNR1B gene expression in the pancreas and glucose and insulin levels in type 2 diabetic rats. koomesh. 2017; 19 (1):46-55.
19. Jorge ML, de Oliveira VN, Resende NM, Paraiso LF, Calixto A, Diniz AL, et al. 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. 2011; 60(9):1244-52.
20. Wei SS, Liang DD. [The effect of exercises on TNF-alpha, IL-6 and adiponectin in different fat diet rats]. Zhongguo Ying Yong Sheng Li Xue Za Zhi. 2013; 29(3):280-2.
21. Glans F, Eriksson KF, Segerström A, Thorsson O, Wollmer P, Groop L. Evaluation of the effects of exercise on insulin sensitivity in Arabian and Swedish women with type 2 diabetes. Diabetes Res Clin Pract. 2009; 85(1):69-74.
22. Bourebaba L, Marycz K. Pathophysiological implication of fetuin-A glycoprotein in the development of metabolic disorders: a concise review. Journal of Clinical Medicine. 2019; 8(12):2033.
23. Salama ME, El-Damarawi MA. Effect of two exercise varieties on fetuin-A plasma level in experimental diabetic nephropathy. Tanta Medical Journal. 2017; 45(1): 21-8.
24. Malin SK, Mulya A, Fealy CE, Haus JM, Pagadala MR, Scelsi AR, et al. Fetuin-A is linked to improved glucose tolerance after short-term exercise training in nonalcoholic fatty liver disease. J Appl Physiol (1985). 2013 Oct 1; 115(7):988-94.
25. Blüher M, Rudich A, Klöting N, Golan R, Henkin Y, Rubin E, et al. Two patterns of adipokine and other biomarker dynamics in a long-term weight loss intervention. Diabetes Care. 2012; 35(2):342-9.
26. Choi KM, Han KA, Ahn HJ, Lee SY, Hwang SY, Kim BH, et al. The effects of caloric restriction on fetuin-A and cardiovascular risk factors in rats and humans: a randomized controlled trial. Clin Endocrinol (Oxf). 2013; 79(3):356-63.
27. Jung CH, Kim BY, Kim CH, Kang SK, Jung SH, Mok JO. Associations of serum fetuin-A levels with insulin resistance and vascular complications in patients with type 2 diabetes. Diabetes and Vascular Disease Research. 2013; 10(5), 459-67.
28. Hennige AM, Staiger H, Wicke C, Machicao F, Fritsche A, Häring HU, Stefan N. Fetuin-A induces cytokine expression and suppresses adiponectin production. PloS One. 2008; 3(3); 1765.
29. Bhattacharya S, Kundu R, Dasgupta S, Bhattacharya S. Mechanism of lipid induced insulin resistance: An overview. Endocrinology and Metabolism. 2012; 27(1); 12-9.
30. Kumar KG, Zhang J, Gao S, Rossi J, McGuinness OP, Halem HH, et al. Adropin deficiency is associated with increased adiposity and insulin resistance. Obesity (Silver Spring). 2012; 20:1394-402.
31. 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 Metab. 2008; 8:468-81.
32. Azam Ramezankhani, Rahman Soori, Aliasghar Ravasi, Ali Akbarnejad. An Investigation of the Effects of Long-Term Exercise on Serum Adropin Levels and Improvement of HOMA-IR in Obese Sedentary Women. Sport Biosciences. 2018; 11(3): 239-51.
33. Sayın O, Tokgöz Y, Arslan N. Investigation of adropin and leptin levels in pediatric obesity-related nonalcoholic fatty liver disease. J Pediatr Endocrinol Metab. 2014; 27(5-6):479-84.