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Effect of 8weeks of low-intensity continuous training on plasma adipolin, insulin resistance, and weight of fatty fat-filled rats

Abstract

Introduction: The purpose of the present study was to investigate 8weeks of low intensity continuous training (LICT) on plasma adipolin, insulin resistance, and high fat obese male rat's weight.

Materials and methods: In this study, 14 male Wistar rats who ate 8weeks of high-fat diet were selected. Six rats were selected as control group for obesity and eight for the control group. Continuing training group, 5 sessions per week and for 8weeks, went on to work on the tape. 24hours after the end of the training session, a blood sample was taken and the levels of adipolin, insulin and plasma glucose were measured. The weight of the rats was also measured every week. For statistical analysis of the findings, independent t-test was used by SPSS-20 software. A significant level of 0.05 was considered.

Results: Data analysis indicated that plasma levels of adipolin in the training group were significantly higher than the control group (p=0.000). Insulin resistance index decreased significantly in exercise group compared to control group (p=0.02). The weight of rats in the training group was significantly lower than the control group (p=0.001).

Conclusion: The results indicated a significant increase in plasma adipolin levels in the continuous training group compared with the control group and possibly with this increased inflammatory activity of the macrophages in the adipose cells and the fat content of the body followed by obesity would be moderated.

Keywords: adipoline, low-intensity continuous exercise, obesity, insulin resistance

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Introduction

In recent years, the rates of obesity and related illnesses have increased in the country, with 71% of Iranian women and men suffering from overweight and obesity.[1] The epidemic of obesity and the prevalence of disorders and associated illnesses have led to an increase of 30% in health care and obesity costs in obese people compared to normal weight counterparts.[2] Therefore, harm to individual health and quality of life has led researchers to study the causes and treatment of obesity as an effort to research their research. Although calorie restriction and dietary therapy are one of the main therapeutic interventions in controlling weight and obesity, exercise training with a 20-80 percent reduction in risk appetite in preventing and reducing the effects of pathologic abnormalities and improving the quality of life plays a role Have.[3] Recent studies have shown that physical activity and exercise may be due to the effect on body fat content and its secretion half-life in Reduce the risk of heart and metabolic diseases. The adipose tissue, as an active and active paracrine tissue, is involved in the synthesis and secretion of a series of hormones and adipocytokines, such as leptin, adiponectin and visfatin, not only in controlling body weight balance, but also by affecting the metabolic and inflammatory profile , Justifies the relationship between overweight and obesity with insulin resistance and diabetes.[4,5] In between, Leptin enhances insulin resistance with its proinflammatory function.[6] Adiponectin is an anti-inflammatory cytokine with anti-diabetic function,[6] and visfatin also contributes to improving insulin sensitivity with its insulin-like role.[7] Often, other adipocytokines have been identified that contribute to modulating insulin resistance. Adipolin is one of these adipocytokines. Adipolin with Adipose-Derived Insulin-Sensitizing Factor (AF) is a 12th member of the family of proteins associated with C1q/TNF-related protein (CTRP), which according to its performance by Enomoto et al.[5] The name was introduced in 2011.[5] Adipolin (CTRP12), as adiponectin, anti-inflammatory cytokine It is mainly synthesized and released in adipose tissue and reduced in obesity, diabetes and other pathological conditions due to obesity.[5,8] Additionally, adipolin also helps to improve insulin sensitivity;[5,8] so that adipolin not only penetrates insulin-dependent pathways, but also improves insulin signalling in adipose tissue and the liver improves insulin resistance, but it also helps with insulin resistance, glucose uptake and insulin secretion following a promise[8,9] through insulin-free pathways. Adipolin was found intact in intact forms (fCTRP12) (40kDa) and broken (spherical) (gCTRP12) (25kDa) in circulation.[8] Studies have shown that only fCTRP12 is an isoform of adipolin, which can improve insulin resistance by activating the pathway of Akt and increasing insulin-induced glucose uptake. Although the other adipolinar isoform, gCTRP12, triggers this pathway by phosphorylation of MAPK, but in improving resistance Insulin does not play.[9] Therefore, any factor that affects the expression of the gene and the synthesis of adipolin, or the breakdown of adipolin and the reduction of its intact form can reduce insulin sensitivity, as well as insulin. Although insulin expresses both forms of adipolin in adipose tissue,[8] it seems to further break the

fCTRP12 and thereby increase gCTRP12.[9] So, decrease insulin levels may be one of the effective ways to improve adipolin function. A lot of ways to reduce insulin in obesity and insulin resistance are known. Physical activity is one of the most important factors. Therefore, it is possible that physical activity and exercise training will not only be directly affected by insulin and improve its performance, but also by influencing adipoline levels and changes in Insulin improves insulin resistance by influencing adipolin levels and changing the ratio of intact intracellular adipolin isoforms to improved insulin resistance. But according to our knowledge, in any of the studies, the effect of exercise training on adipoline levels has not been studied. Therefore, considering the key role of adipoline in modulating insulin resistance along with obesity, effective protocol selection is necessary. The American Diabetes Association is implementing a minimum of 150minutes of moderate-intensity aerobic exercise, three days a week for weight loss, recovery Control glucose and reduce the risk of cardiovascular disease.[10] Therefore, assuming that the use of continuous exercise intervention can contribute to the improvement of obesity and metabolic half-life, the present study seeks to answer the question whether low continuity training on serum adipoline, insulin, fasting glucose, and resistance index Does insulin have a significant effect on obese male rats?

Materials and methods

The present study was an experimental design with a post-test design with control group that was carried out in the spring of 1993 at the University of Tehran Animal Hospital. For this purpose, 14 Wistar male rats were purchased from Pasteur Institute of Iran at 6weeks and weighing 110±10g. They were transferred to the animal house of the Faculty of Physical Education and Sports Sciences of Tehran University in accordance with the policy of the Iranian Association for the Protection of Women the lab animals were used for scientific and laboratory purposes. The all of rats under controlled environmental conditions with an average temperature of 22±22°C, a dark-blue cycle of 12:12hours, a relative humidity of 50%, and free access to water and special food Mice were kept in 4-cage cages. In order to adapt to the new environment, they were kept in their cages for 2weeks without any intervention, and during this time, the steadfast diet was fed. After familiarity and adaptation to the new environment, rats were placed under a high-fat diet for 8weeks (prepared by the Razi Serum Institute), which contained 45% of the total energy of fat (derived from animal fat) Which contains 24grams of fat, 24grams of protein and 41grams of carbohydrates per 100grams.[11] During this period, healthy control rats had Free to the standard food. The rats were divided into 2 groups (6 head) and 8 endurance training. Also, four other rats were selected as a pilot group for measuring treadmill's maximum running speed. To estimate the running speed, we performed a graded sport performance with a zero-degree gradient that started at 10m/min and the treadmill speed was increased by 1m/min for 1minute so that the rats could not run (exhaustion).[12] After estimating the maximum speed, the endurance training rats received 5 sessions per week for 8weeks on the drill. Before the beginning of the training, 1week the rats were introduced with a treadmill and run on it at a speed of 5 to 10m/min for 10minutes with a 0 degree gradient. After the introduction, the protocols read the training started at a speed of 15m/min for 25minutes, and was followed by intensive training for 3minutes perminute and 1 meter perminute for observance of the principle of overloading, so that during the final week of the training period it was 50minutes and Training speed reached 22meters perminute. Also, 10minutes of warm-up at the beginning of each session and 5minutes of cool-down at the end of each session were conducted at low intensity. It should be noted that all biological conditions for the control group, other than the practice protocol, were similar to the training group and for the observance of the cases in the present study, sound was used to stimulate activity, and no wind shock or electric stimulation was used. In order to collect the samples, the experimental groups were anesthetized intraperitoneally for 48hours after the last exercise session with a combination of ketamine (75mg/kg) and xylsein (10mg/kg). After ensuring animal anesthesia, the chest of the animal was split and about 10 ml of blood was taken directly from the animal's heart. For separation Plasma samples were centrifuged for 15minutes at a speed of 3,000rpm and transferred to liquid nitrogen and transferred to a freezer temperature of -80°C for further measurements. Adipolin plasma was measured using the family of gene sequencing kit 132 similarity 132, member A (FAM132A), an ELISA Kit from My Bio M Sourcse Inc., USA. The sensitivity of the measurement was 0.1ng/ml. To measure plasma glucose concentration by glucose oxidase enzyme-colorimetric method using glucose kit (Pars Test, Iran) Size Made. The coefficient of variation and sensitivity of the measurement method were 1.8% and 5mg/ml. Plasma insulin measurement was performed by ELISA method using Mercodia Rat Insulin ELISA kit (constructed in Sweden) with sensitivity of 0.07µg/dl. The index of insulin resistance was also calculated by the HOMA-IR method using the following formula: 405/Fasting insulin (micro unit per ml) Fasting glucose (mg/dL)= Hma formula (insulin resistance index).

Statistical analysis

The data were analyzed by SPSS software version22 at the significance level (P≤0.05). After the data were normalized by Kolmogorov-Smirnov test (K-S), independent t-test was used to determine the significance of the differences between the groups.

Findings

Independent t-test results for the variables measured in the present study are presented in Table 1. Analysis of data related to body weight, plasma insulin, plasma glucose and insulin resistance index showed a significant difference between the groups. Body weight (P=0.001) of plasma glucose (031 (P=0.009) and insulin resistance index (P=0.02) in the continuous exercise group were significantly lower than the control group (Table 1). Also, independent t-test results for Plasma adipoline levels showed that after 8weeks of low-intensity continuous training, adipoline levels were significantly higher than the control group (P=0.000) (Figure 1). Further, the weight of rats and glucose, insulin and insulin resistance levels in the training group were shown in Figures 2-5 in the training group.

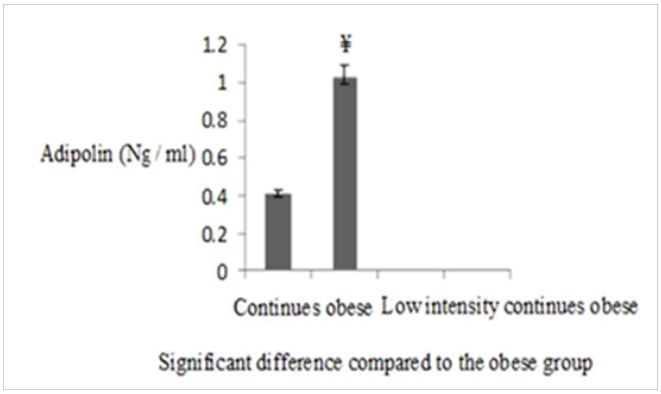

Figure 1 Plasma adipoline surface changes in training and control groups.

Table 1 Independent t-test results for adipolin weight, glucose, insulin and insulin resistance between training and control groups

Training	Low intensity continuous training	Obese control	t	p
Adipolin (Ng/ ml)	1.042±0.13*	0.4181±0.04	10.52	0.000*
Weight (g)	322.37±15.28*	417.5±16.95	9.84	0.001*
Glucose (mg/ dl)	123.82±13.93*	144.28±17.48	2.44	0.031*
Insulin (micro unit bpm)	9.38±0.34*	9.08±0.51	3.08	0.009*
Insulin resistance index	2.57±0.38*	3.25±0.56	2.68	0.02*

*Numbers are expressed as mean±standard deviation

*Significance compared to the control group

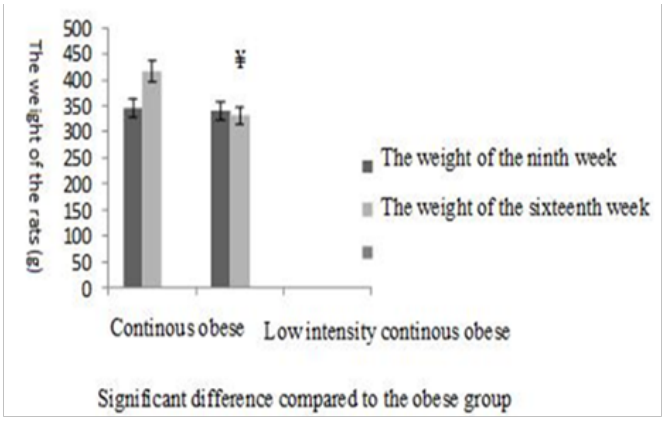

Figure 2 Weight variation of rats in the 9th and 16th week of training in exercise and control groups.

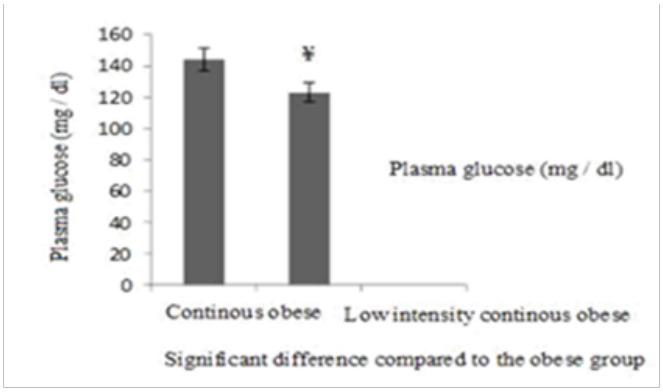

Figure 3 Plasma glucose changes in training and control groups.

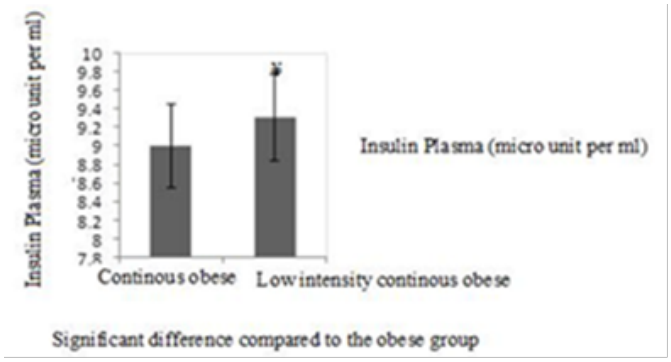

Figure 4 Insulin changes in training and control groups.

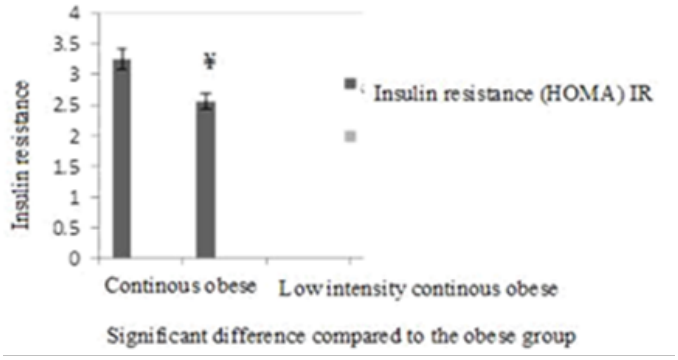

Figure 5 Insulin resistance changes in training and control groups.

Discussion

According to the results of this study, the implementation of 8weeks of low-intensity continuous training increased serum adipolin in the experimental group and this change was statistically significant (p=0.0001). Since no studies have evaluated the effect of any type of exercise or exercise on adipolin levels, the researcher, based on the theoretical foundations and effective factors regulating gene expression and serum adipolin levels, justifies the changes in adipolin after running 8weeks of low-intensity continuous training. Gene expression and serum adipolin levels in obese humans and animals decreases.[5] In fact, adipolin expression is under the negative control of obesity-related stress, so that, by inducing TNF-α and endoplasmic stress in the adipose cell culture medium, adipolin expression declines.[5] TNF-α, including anti-inflammatory adipocytokines derived from adipose tissue and negative regulator adipolin[5] is sought after low intensity exercise and weight loss.[13] TNF-α is capable of affecting some of the translation factors affecting metabolism on adipolin levels; KLF-15 is one of these factors. KLF-15 is a member of the large family of KLF transcription factors that contribute to the regulation of glucose metabolism and adipogenesis.[14,15] Enomoto and colleagues As adipolin, the expression of KLF-15 is also less in the adipose tissue of the DIO mice than in the control group,[16] and the induction of inflammatory conditions and the use of TNF-α decreases the adipolin and KLF-15 mRNA levels in adipose tissue cells.[16] Since TNF-α activates JNK in adipocytes, it increases the expression of proinflammatory cytokines, and, with the exacerbation of inflammatory conditions of adipose tissue,[17] increases the resistance Insulin due to obesity,[18–20] it has been suggested that TNF-α activates JNK by reducing the expression of KLF-15 and subsequently reducing the expression of adipolin in fat cells,[16] and thereby Provides or exacerbates insulin resistance. Endoplasmic Endotracheal Stress

(ER) is also an inflammatory factor that interacts with obesity and inflammatory conditions.[21] And expression of adipolin in the cell Reduces the crop fat.[5] ER stress under conditions associated with obesity and type II diabetes is over-activated in many tissues and helps spread inflammation, apoptosis of pancreatic beta cells, insulin synthesis disorder and insulin resistance.[21] The ER response, also called the Uncritical Protein Response (UPR), is the response of the endoplasmic endothelial network to ER stress to match the functional capacity of the endoplasmic network to cellular demand and to improve these disorders due to ER is stress.[21] Since exercise is one of the proposed therapies for the improvement of inflammation and the prevention and treatment of obesity and metabolic disorders associated with it, such as type II diabetes and insulin resistance, some studies aimed at identifying intermediary molecular mechanisms in the effects of physical activity and Exercise In this chapter, we examine the effects of different types of exercise protocols on TNF-α and ER-related molecules. Studies have shown that TNF-α levels follow the exercises Continuous low weight and weight loss is reduced.[13] Additionally, compatibility with sport exercises, while improving the inflammation of inflammation in obese subjects, inhibits ER stress and improves UPR.[22] Although the present study did not measure the levels of TNF-α and biomarkers associated with ER or UPR, however, due to the relationship between TNF-α and biomarkers associated with ER or UPR stress with changes in obesity, it is expected to reduce weight significantly, Body mass index and body fat percentage after 8 Low Tensile Continuing Exercises In this study, TNF-α levels and ER stress decreased, and subsequently plasma adipoline levels increased in the training group. However, due to the lack of alignment of adipoline plasma levels with changes in body weight after 8weeks of training, non-obesity is likely to affect adipolin levels after exercise, which is less likely to be affected by weight loss following exercise exercises. And improve the distribution of body fat;[23] insulin can be one of these candidates.[23] Insulin is the regulator of carbohydrate metabolism and glucose hemostasis, by binding to the alpha-receptor subtype of insulin receptor and triggering insulin signal pathway increases the transfer of GLUT4 to the cell membrane and transfusion tubes, and increases glucose clearance.[24] The regulation of insulin is based on dipyridine, so that in the lean subject, activating the PI3K pathway can increase the expression and secretion of adipoline,[25] but in obesity that is also susceptible to insulin resistance, this is a haemostatic interaction insulin and adipolin interact and insulin reduces the levels of adipoline.[26] In this study, plasma glucose levels and HOMA-IR values after 8weeks of low continuity training change was statistically significant (p=0.031, p=0.02). Exercise exercises through:

a. Increased insulin receptor.

b. Increased protein and mRNA carriers of glucose (GLUT4).

c. Increased glycogen synthase, protein kinase-B and hexokinase.

d. Improved intracellular insulin messaging and effects on intermediate molecules in Insulin signal such as increased expression of ERK2, increased activity of PI3K or Akt / PKB, and improved AMPK signal.[27]

e. Changes in muscle composition (increased capillary density in muscle fibers and conversion Muscle fibers to fast oxidizing contraction fibers.

f. Increase glucose delivery to muscle.

g. Decrease the accumulation of triglycerides in muscle cells.

h. Reduce the release of fatty acids and increase oxidation and purify them,[28] modulates insulin resistance.

The two characteristics of the severity and duration of the practice of insulin response to exercise are strongly influenced, so that the improvement of insulin sensitivity occurs when the volume of exercise is at its highest.[29] Since the subjects of the present study are rats. The boys were obese and there was no limitation in the design of the training features. It can be said that the intensity, duration and volume of exercises were suitable for modifying the levels of insulin, glucose and HOMA-IR by any of the above pathways. They are regarding the inverse relationship between insulin and glucose with adipolin, a significant change in insulin and glucose can be one of the causes of a significant change in adipolin after 8weeks of low continuation training.

Conclusion

The results showed a significant increase in plasma adipolline levels in the continuous exercise group compared to the control group and possibly with this increased inflammation of macrophages in fat cells and the body fat and subsequent obesity were also reduced. Since this study is one of the first researches on the effect of aerobic exercises on plasma adipolin levels, more studies are needed to understand the interface mechanism.

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None.

Conflict of interest

The author declares no conflict of interest.

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