Effect of 8 weeks 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 8 weeks 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 8 weeks 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 8 weeks, went on to work on the tape. 24 hours 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

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. 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. 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. 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. In between, leptin enhances insulin resistance with its proinflammatory function. Adiponectin is an anti-inflammatory cytokine with anti-diabetic function, and visfatin also contributes to improving insulin sensitivity with its insulin-like role. 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. The name was introduced in 2011. 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. Additionally, adipolin also helps to improve insulin sensitivity; so that adipolin not only penetrates insulin-dependent pathways, but also improves insulin signaling in adipose tissue and the liver improves insulin resistance, but it also helps with insulin resistance, glucose uptake and insulin secretion following a promise through insulin-free pathways. Adipolin was found intact in intact forms (ICTRP12) (40kDa) and broken (spherical) (gCTRP12) (25kDa) in circulation. Studies have shown that only gCTRP12 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. 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, it seems to further break the
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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 6 weeks 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±2°C, a dark-blue cycle of 12:12 hours, 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 2 weeks without any intervention, and during this time, the steadfast diet was fed. After familiarity and adaptation to the new environment, rats were introduced with a treadmill and run on it at a speed of 5–10 m/min. 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 15 minutes at a speed of 3,000 rpm and transferred to liquid nitrogen and transferred to a freezer temperature of -80°C for further measurements. Adipolin plasma was measured by the family of gene sequencing kit 132 similarity 132, member A (FAM132A), an ELISA Kit from My Bio M Source 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/ml. The index of plasma insulin 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 version 22 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 (0.01 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 8 weeks 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.

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**Figure 1** Plasma adipoline surface changes in training and control groups.
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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*                       | 0.418±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

Discussion

According to the results of this study, the implementation of 8 weeks 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 8 weeks of low-intensity continuous training. Gene expression and serum adipolin levels in obese humans and animals decreases. 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. TNF-α, including anti-inflammatory adipocytokines derived from adipose tissue and negative regulator adipolin is sought after low intensity exercise and weight loss. 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. 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, and the induction of inflammatory conditions and the use of TNF-α decreases the adipolin and KLF-15 mRNA levels in adipose tissue cells. Since TNF-α activates JNK in adipocytes, it increases the expression of proinflammatory cytokines, and, with the exacerbation of inflammatory conditions of adipose tissue, increases the resistance of insulin due to obesity. 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, and thereby provides or exacerbates insulin resistance. Endoplasmic Endotracheal Stress
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. 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 8 weeks of low continuation training.

Conclusion
The results showed a significant increase in plasma adipolin 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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Conflict of interest
The author declares no conflict of interest.

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