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The Effects of Three Months of HIIT on Plasma Adiponectin on Overweight College Men

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***Abstract*—**Adiponectin is a cytokine secreted by the adipose tissue that functions as an anti-inflammatory, antiathrogenic and anti-diabetic substance. Its density is inversely correlated with body mass index. The purpose of this research was to examine the effect of 12 weeks of high intensity interval training (HIIT) with the level of serum adiponectin and some selected adiposity markers in overweight and fat college students. This was a clinical research in which 24 students with BMI between 25 kg/m^2 to 30 kg/m^2. The sample was purposefully selected and then randomly assigned into two groups of experimental (age =22.7±1.5 yr.; weight = 85.8±3.18 kg and height =178.7±3.29 cm) and control (age =23.1±1.1 yr.; weight = 79.1±2.4 kg and height =181.3±4.6 cm), respectively. The experimental group participated in an aerobic exercise program for 12 weeks, three sessions per weeks at a high intensity between 85% to 95% of maximum heart rate (considering the over load principle). Prior and after the termination of exercise protocol, the level of serum adiponectin, BMI, waist to hip ratio, and body fat percentages were calculated. The data were analyzed by using SPSS: PC 16.0 and statistical procedure such as ANCOVA, was used. The results indicated that 12 weeks of intensive interval training led to the increase of serum adiponectin level and decrease of body weight, body fat percent, body mass index and waist to hip ratio ($P < 0.05$). Based on the results of this research, it may be concluded that participation in intensive interval training for 12 weeks is a non-invasive treatment to increase the adiponectin level while decreasing some of the anthropometric indices associated with obesity or being overweight.

***Keywords*—**Adiponectin, interval, intensive, overweight, training.

I. Introduction

OBESITY is a risk factor for affliction to many diseases that result in morbidity and mortality in industrial, and developing countries as well. Obesity is part of a disease known as the metabolic syndrome which is a metabolic disorder. With the increase of body fat percent, accumulation of lipids around the visceral area occurs [1]. In sedentary persons, obesity and increase in body weight occurs. There is a close association between chronic disease such as hypertension and body fat and resistance to insulin, all of which are the known risk factors for cardiovascular disease [2].

Recently adipose tissue is identified as an active paracrine and endocrine organ that synthesis and secrets a collection of adipocytokine and active bio mediator such as adiponectin that not only plays a role in the control of body weight but also has an effect on the lipid profiles, metabolic and inflammatory processes explaining the association between the obesity, overweight and resistance to insulin, diabetes and atherogenic cardiovascular disease [3]. Adiponectin is secreted by the adipose tissue as an adipokine and plays as significant role in regulating energy needed for maintaining hemostasis, metabolism of lipids and carbohydrates [4]. The density of adiponectin is between 5 to 30 micro grams per liter and make up approximately 1% of total plasma. Adiponectin hormone contains 244 amino acids in three forms of high, medium and low molecular weight in circulation [5], [6]. Contrary to the other cytokines originating from adipose tissue, adiponectin has anti-inflammatory, antiatherogenic and anti-diabetic effect [7] Through controlling the synthesis of TNF-α and IL-6 and as a result CRP, it controls the inflammatory responses and by decreasing the ICAM-1 gene expression and improvement of endothelial function, it decreases the progress of atherosclerosis [8].

The level of adiponectin is inversely correlated with body fat percent. Patients suffering from diabetes, high blood pressure and ischemia heart disease have adiponectin density level less than that of normal healthy persons [9]. In addition, according to the laboratory findings, it has been shown that adiponectin regulates glucose metabolism and insulin sensitivity through the activation of AMP kinas. In addition, to the regulation of glucose homeostasis and fat metabolism, adiponectin is associated with cardiovascular fitness and health. The existing evidence shows that reduction in adiponectin in circulating blood is correlated with the prevalence and the intensity of atherosclerosis [10], [11].

The present evidence indicates that the decrease in the level of adiponectin in blood circulation is associated with the intensity of atherosclerosis [11].

Contrary to the other adipocytokines, the expression of mRNA and the amount of adiponectin in fat, diabetes, and coronary ill individuals is less and increases with body weight loss [12]. Hypo adiponectinomy can lead to the resistance to insulin and increase of affliction to diabetes type 2. These conditions are likely to present a new risk factor for coronary heart disease [13], [14]. Therefore, this late marker has attracted attention from the sport medicine and exercise science experts. They have introduced protocols to examine

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different forms of exercise on this marker. Rashid et al. have shown a significant increase in adiponectin level (P<0.05) following the participation in aerobic exercise [15].

Hemmati et al. reported that six weeks of high intensity exercise resulted in significant increase in adiponectin level [16]. Kraemer et al. reported a decrease in the level of plasma adiponectin level following the participation in a sufficient intensity exercise [17]. Trapp et al. reported that interval training at a high intensity level lasting more than two months compared to the resistance training resulted in a significant decrease in body fat percent, weight and BMI in sedentary young women [18]. Kordi et al. demonstrated that high intensity exercise resulted in significant increase in adiponectin levels and decrease in BMI, weight, body fat percent, and waist to hip ratio (WHR) in sedentary young women [19]. On the other hand, Buchan et al. and Metcalf et al. also reported the adiponectin level decreased as a result of participation in physical activity [20]-[22].

Such contradictory findings and limited number of research in regard to the effect of intensive exercise on the level of adiponectin change and the significance of the subject for decreasing the risk of cardiovascular and other disease makes this a subject of interest to examine more closely. Thus, this research was designed to determine the effect of high intensity exercise on the level of adiponectin in sedentary overweight and obese young male participants.

II. Material and Methods

This was a clinical trial in which 24 volunteer overweight or fat students at the University of Isfahan were purposefully selected. The procedures were explained to the participants and following the signing of the human consent form, they were randomly assigned to two groups of (n=12) experimental (mean for age 22.7±1.5 yr, weight= 85.8±3.18 kg, height= 178.7±3.29 cm) and control (mean for age 23.1±1.1 yr, weight= 79.1±2.24kg, height=181.3±4.6 cm). The subjects in the experimental condition completed a shuttle ran of 40 meters distance at their highest possible speed.

The subjects performed this task for 30 seconds inter spaced by 30 seconds of rest intervals, four times in the first and second weeks. The frequency was increased to five and six times in the third and fourth week and continued to increase to eight times until the eighth week [23]. Warm-up and cool down exercises were performed at every session.

The subject in the control group did not participate in the exercise protocol and engaged only in their regular activities. The inclusion criterion was set to a BMI ranging between 25 kg/m^2 to 30 kg/m^2, having no history of regular physical activity within the last six months, having no serious medical history or use of medication at the time of protocol.

The subjects attended a lab and 5 cc of fasting blood sample was collected before the start of the exercise program and 48 hours post the termination of the exercise protocol. The blood samples were collected at 8 a.m. and plasma serum was separated to be kept at ─ 80 °C. The adiponectin level was measured by ELISA method using Adiponectin kit made by South Korea (Adipogen South Korea) with the sensitivity of 0.1 µg/ml. The coefficient of variation of this kit for external test was 3.9% and the internal variation coefficient was 8.6%. The percentage of fat was measured by electronic adipometer Omron set using Cash brothers-three-site method [24]. BMI was calculated by dividing weight in kilograms to the square of the height in meters, and WHR was measured by flexible tape and dividing the waist size to the hip measure. Statistical analysis was performed by using SPSS: 16.0 and ANCOVA was employed to test the hypothesis at alpha level set to 0.05.

III. Results

The result of analysis indicated that 12 weeks of HIIT resulted in significant increase of plasma adiponectin (P<0.001), reduction of BMI (P<0.001), body weight (P<0.001), body fat percent (P<0.001) and WHR (P<0.001). These results are presented in Table I. The experimental group compared to the control group showed a reduction of BMI (P<0.001), body weight (P<0.003), body fat percent (P<0.001) and WHR (P<0.001), while the adiponectin level increased (P=0.001). The size of difference in the experimental group was 61.1% increase in adiponectin, 8.1% decrease in BMI, 7.7% in weight, and 16.6% decrease in body fat and 5.05% decrease in WHR.

TABLE I
Comparing the Mean and Standard Deviation of Adiponectin, BMI, Body Weight, Body Fat Percent, WHR in Pre- and Post-Test Condition

Groups	Experimental		Control	
Variables	Pre-test Mean ± SD	Post-test Mean ± SD	Pre-test Mean ± SD	Post-test Mean ± SD
Adiponectin level µg/ml	0.47 ±16.71	#*1.08 ± 20.73	0.15 ± 17.12	0.16 ± 17.10
BMI kgm2	1.4 ± 26.29	#*1.03 ± 24.7	1.7 ± 24.95	1.54 ±26.14
Body weight kg	#3.18 ± 85.8	*#2.4 ± 79.18	3.9 ± 82.6	2.8 ±82.9
Body fat percent	2.13 ± 31.9	*#2.22 ± 26.6	1.7 ± 30.3	1.54 ±30.2
WHR	0.015 ± 0.99	*0.045 ± 0.94	0.019 ± 0.98	0.022 ±0.97

* Sig at 0.05 pre-post test # Experimental vs. Control

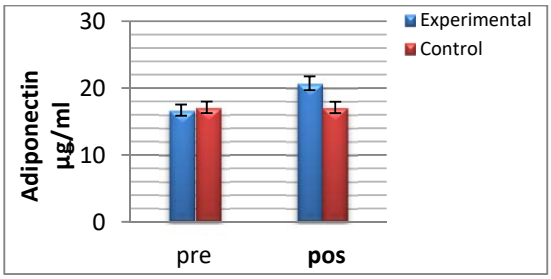

Fig. 1 Mean of pre and posttest in experimental and control group

IV. Discussion

Recently, interest to understanding the mechanisms involved in pathophysiology of obesity and weight gain, particularly metabolic disorders, has increased considerably [24]. At the first inspection, it seems like a very simple mechanism control this process, however, a closer look at the subject reveals that very complicated processes occurring at

the cellular level are very important. The results of the research conducted to examine the mechanism related to the occurrence of obesity and the incidence of cardiovascular disease has shown that the increase in body fat mass is associated with the incidence of the disease [25]. The results of the analysis in this research indicated that 12 weeks of HIIT resulted in significant increase of plasma adiponectin, reduction of BMI, body weight, body fat percent and WHR in the experimental group.

This result is similar to what was reported by Hemati and associate (2013), Kordi and associates (2013), Rashidlamir and associates (2013) and Trapp (2008) [15], [16], [18], [19]. Considering the results of the present research, physical exercise has been shown to increase the absorption of glucose in peripheral tissues in response to acidosis, accumulation of lactate, increase in sympathoadrenal presence, energy homeostasis, glycogen depletion, and glycolysis control improves insulin sensitivity and leads to the increase in plasma level [26]. Since the concentration of plasma fatty acid has a positive effect on adiponectin secretion, it is likely that exercise increases the rate of lipolysis in fat tissue and the level of plasma fatty acid and cholesterol is increased that eventually causes intercellular adiponectin level [27].

There are reports claiming that the more calories spent during exercise and more body is stressed for metabolism and adaptation, the more Adiponectin is needed to regulate the metabolism processes and the more Adiponectin is secreted [28]. Such condition was confirmed by the results of this research.

It is known that cAMP is generated in adipose tissue and the liver with the help of adenylate cyclase enzyme. cAMP activates phosphorylase and the process of golycolysis on the one hand, and lipolysis on the other [29]. Perhaps the reduction of body weight and fat accumulation in this research was due to this process. It needs to be mentioned that such a mechanism is involved in an increase in the activity of glucose carriers (GLUT4) that occurs during high intensity activity that leads to the entry of glucose to adipose tissue through GLUT4 presence; thus, causing the extraction of glucose from the adiposities and regulate insulin sensitivity in adipose tissue [28].

Therefore, one of the possible explanations for serum adiponectin increase following 12 weeks of high intensity exercise is the decrease in weight and improvement of body composition, as was the case in this study. In addition, there are evidences that show physical exercise causes an increase in the production of mitochondria in white fat tissue that finally results in a decrease in body mass index, WHR and increase of plasma adiponectin [30]. Such an assumption however remains as speculation and needs further investigation.

Another possible explanation in regard to the effect of high intensity interval exercise on the increase of serum plasma and decrease in body weight, BMI, body fat percent in this research, is the fact that the large proportion of fatty acid needed for the muscular contractions during physical activity is provided by three- to four-fold increase in lipolysis of glycerides in fat tissue. High intensity exercise leads to a two-fold increase in blood circulation to fat tissue and leads to a 10 fold increase in circulation to the active muscles. Many researchers claim that the decrease in body fat, WHR, and body composition is due to the imbalance of input-expenditure of energy and production of negative electricity which may lead to the increase in the level of adiponectin and decrease of plasma leptin following participation in exercise [31]. It appears that all the needed factors were present in this high intensity exercise to increase the level of serum adiponectin. On the other hand, the results of this study are not in agreement with the findings reported by Buchan et al., Platat et al. and Metcalf et al. [20]-[22]. There are reports that claim the increase in physical activity results in an increase of adiponectin receptors, which in turn causes a decrease in adiponectin level [32]. It has also been stated that the release of adiponectin may decrease during the time when the exercise is suitable for the improvement of blood sugar in normal individuals [20].

The contradictory results in regard to the level of change in adiponectin may be attributed to the different time of drawing the blood sample, types of exercise protocol, and the subjects participating in the study [20], [33]. However, the subject needs further investigation. It seems that 12 weeks of HIIT can increase the level of serum adiponectin, and decrease adiposity indices related to extra body weight. The sample size used in this research was one of the limitations; however, the researcher tried to recommend the participants to maintain a diet given by a dietitian. The participants were students residing in the dormitory of the university and it was possible to present a similar diet for all of them. However, complete control over the entire sample was not feasible.

V. Conclusion

The results of this research indicate that HIIT increases the level of plasma adiponectin; decreases body fat percent, weight, BMI, and WHR of overweight male students. Therefore, it can be concluded that high intensity exercise protocols can serve as a means by which the level of serum adiponectin is regulated and function as a method to prevent or postpone diseases associated with overweight and obesity; as well as being a noninvasive strategy to control or decrease the risk of affliction to inflammatory disease, and the likelihood of metabolic syndrome.

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