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Effect of aerobic training on insulin resistance and plasma adiponectin in type 2 diabetes women

Behbudi Laleh*, Hajirasouli Masoud, Afsharmand Zohreh

Department of Physical Education and Sport Science, Islamshahr Branch, Islamic Azad University, Islamshahr, Iran

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Abstract

Over the past two decades, the global prevalence of diabetes has substantially increased. Aerobic exercise has identified as a specific treatment and major strategy for treatment of patients suffering type-2 diabetes. So this study aims to explore the effect of aerobic exercise on concentration of plasma adiponectin and insulin resistance in women with type-2 diabetes. 20 diabetic women were placed as subjects into aerobic exercise and control groups. The aerobic exercise program included three weekly walking sessions for 30 to 60 minutes with the intensity of 60 to 70 HRR. Plasma adiponectin concentration, fasting glucose, insulin, blood lipids and body measurements were measured at the beginning of the program (pre- test), fourth week (during the test) and at end of the sixth week (post-test). Independent t test was used to analyze the data and the two-way analysis of variance was made using repeated measurements. After 8 weeks of exercise, plasma adiponectin concentration and insulin resistance did not represent any significant change ($P>0.05$), but fasting glucose represented a significant decrease ($P<0.05$). The findings of this study showed that 8 weeks of aerobic exercise would not have a significant influence on plasma adiponectin and insulin resistance in type 2 diabetic women; however, it had a significant effect on weight, BMI and fasting glucose level of this group of diabetic women. In addition, 8 weeks of aerobic exercise brought about a significant impact on VO2max. Therefore on the basis results from this study it is recommended that type-2 diabetes able to perform aerobic exercises should benefits from the useful effects of this type of exercise.

***Corresponding Author:** Behbudi Laleh ✉ behbudi@gmail.com

Introduction

In today's world widespread overweight and obesity due to sedentary lifestyle in industrialized societies and the availability of machineries required for modern life, the number of people susceptible to Type 2 diabetes is increasing day by day and this condition will be one of the health concerns of the twenty-first century (Hu *et al.,* 2006). The main cause of type 2 diabetes is that the body cannot produce sufficient insulin and since the body does not respond properly to insulin, a disorder is developed called "insulin resistance" or decreased insulin sensitivity. These abnormalities result in dysfunction in the spending and storage of carbohydrates, increased blood sugar and stimulated compensatory increase in insulin secretion (hyperinsulinemia) and subsequently patients face excessive weight gain and obesity. In these individuals certain peptides secreted by adipose tissue, called adipocytokine the amounts of which are associated with obesity, impact metabolism of glucose and lipid (Pittas *et al.,* 2004; Matsuzawa *et al.,* 2004). Compared to other adipocytikines, plasma adiponectin decreases in obesity and insulin resistance (Hoffested *et al.,* 2004). This is confirmed by a research reporting that circulating adiponectin levels are inversely correlated with body fat percent, insulin resistance and type II diabetes (Yamauchi *et al.,* 2001). Furthermore, adiponectin is identified as the most important factor in the regulation of energy homeostasis and insulin sensitivity playing a fundamental role in metabolism of fat and glucose (Tsuchida *et al.,* 2004).

In recent years, several studies have been conducted on finding appropriate treatment strategies for type-2 diabetes with minimal side effects. Among recent studies are those examining the impact of regular physical activity as one of the main three pillars of controlling type 2 diabetes (1- diet control; 2- medication or timely insulin injections 3- exercise that causes boosted supply of sugar to active muscular tissues and thus lowers blood sugar) on markers of diabetes and contributing factors including adiponectin (Misra *et al.,* 2008; Davenport *et al.,* 2008; Lambers *et al.*, 2008). Aerobic exercise has been identified inter alia a particular treatment and a major strategy for patients with type-2 diabetes (Stephan *et al.,* 2007; Hisayo *et al.,* 2004). However, few studies have been conducted on the effects of exercise on adiponectin and due to inconsistent results no general theory can be derived from them (Hisayo *et al.,* 2004; Ifigena *et al.,* 2005; Luccotti *et al.,* 2006; Bluher *et al.,* 2007). Therefore, given the prevalence of type-2 diabetes and the complications caused by the disease, and since the most important research concepts in modern sports physiology for treatment and control of diabetes, is to study metabolic responses to training methods, including aerobic exercise it is more imperative than ever to study the effects of an aerobic exercise program on insulin resistance and plasma adiponectin in women with type 2 diabetes.

Materials and methods

Subjects

The statistical population of this study consists of all women with type-2 diabetes referring to Karaj City Diabetes Association Clinic who had a medical record at the clinic. After completion of a face-to-face interview and the demographic and medical history questionnaire 20 of the population aged 50-65 years with no chronic and acute diabetes with fasting blood sugar lower than 250 mcg/dL, were selected as subjects. Before getting consent letters from the subjects, the subjects were briefed with necessary information about the nature of the study, manner of conducting the study and the possible risks and points they should observe to participate in the research both in writing and verbally. Then subjects signed consent form. The subjects were reassured that their information would remain confidential and would only be disposed in form of mean and that they were free to stop participating in the study at any time they wished. The subjects did not smoke; nor did they participate in regular exercise. After this phase, the subjects were randomly divided into two groups of ten; aerobic exercise and control groups. The subjects were asked not to change their treatment and medications during the research. Table 1 represents the general characteristics of the subjects.

Table 1. Mean and standard deviation of age and anthropometrical markers of all participants.

Variables	Arobic training group	Control group
Age (year)	55.7 ± 4.76	57 ± 5.72
Height (cm)	70.6 ± 6.04	69.3 ± 7.92
Weight (kg)	161.2 ± 6.97	157.6 ± 7.79
History of diabetes (year)	5.7 ± 3.13	6.7 ± 2.67

Physiological Measurements

BMI in subjects was calculated by dividing the body weight in kilograms by the square of height in meters. Body fat percentage was calculated using calipers HARPENDEN.CO, CE0120 made in England and satiation formula (Donald *et al.,* 2010). Insulin resistance was measured using homeostatic assessment of insulin resistance (HOMA-R) using fasting glucose and insulin concentrations to determine insulin resistance in type 2 diabetic patients (Tso *et al.,* 2006).

Blood sampling

To determine adiponectin, insulin and glucose, 10 cc of blood from was taken from antecubital vein in less than a minute after fastening the tourniquet in sitting position after 12 hours of fasting at the beginning of the research period (pre-test), after 4 weeks (mid-test) and after From 8 weeks (after the test). Then 3 ml of plasma obtained was frozen to measure plasma adiponectin levels in each stage at 80 - ° C to be analyzed at the end of the exercise period. All sampling was done at 8 AM. The subjects were asked to avoid physical activity beyond their routine daily physical activity 24 hours before blood sampling.

Biochemical Measurements

Adiponectin concentration was measured using (Eliza) method with use adiponectin kit made by European Union Biovendor Company with internal degree measurement 6.4 and 26 ng/ml sensitivity. Insulin concentration was measured using (Eliza) method using insulin kit made by Italian Diametra Company with 2% degree of internal measurement and 2 uIU/Ml sensitivity; glucose concentration was measured using GOD Photometric Enzyme Method using Pars-Azmoon Company glucose with degree of internal 1.28 mg, the sensitivity 5 ma/DL.

Aerobic Exercise intervention

Aerobic exercise group participated in an 8-week aerobic exercise program with the frequency of three sessions per week. In the first three weeks for 30 minutes (alternating between 5 minutes running at the intensity of 60-70 % of maximum heart rate reserve and 5 minutes of active rest with 30-45 % of maximum reserve heart rate), in the fourth to sixth week, each session took 42 minutes (alternating between 7 minutes of running with the intensity of 60-70% of maximum heart rate reserve for 30-45 % of maximum heart rate of 7 min rest with storage) and seventh-eighth week of training, 60 minutes (the periodic 10 minutes of running with 70-60% of maximum heart rate reserve for 10 minutes with an intensity of 45-30% of maximum heart rate reserve rest) to aerobic exercise. Each session included a warm up with stretching and rotating joints and slow walking, so the main program involved aerobic exercise program and cooling down by stretching and rotation of the joints and walking. To prevent hypoglycemia and dehydration the participants were asked to drink 100 ml of water and sugar syrup with 5% sugar concentration before each session. All training sessions were performed between 50 to 70 minutes.

Statistical methods

All data resulting from the analysis of blood samples and body measurements obtained from the subjects before and after 4 and 8 weeks are reported on the basis of mean and standard deviation. Mean quantitative variables measured were compared between the two groups before the intervention using independent t test. To compare before and 4 and 8 weeks after the intervention two-way analysis of variance with repeated measurements with the three times measurements for each of the groups

(experimental and control) were used and in the abovementioned analysis due to significance of main effects or interactions of measurements, in their pursuit of measurements ANOVA was repeated for each of the groups. Repeated measurements necessitated establishing two prerequisites of normal distribution of data and the sphericity of the variance-covariance matrix. To examine normal data distribution prerequisite Kolmogorov - Smirnov test was used and the prerequisite was satisfied for all variables.

The second prerequisite was tested during examination of each hypothesis separately using Mauchly Test. Once establish the test procedure was followed and upon establishing the assumed sphericity the results were reported otherwise Greenhouse-Geisser method was used to deal with the problem and the results were duly reported.

Where necessary, the means related to this variable were compared with each other at different times as well as between the groups. For this purpose repeated measure analysis of covariance was performed using Bonferroni and for comparison of means in the groups at separate measuring times analysis of variance was performed using an appropriate post hoc test. The purpose of these analyses was accordingly to find the best time in each of the groups and the best group at each of the times and if interaction between time and group was significant, it aimed at finding the best group - time combination. Analysis of covariance (ANCOVA) was applied to compare quantitative variables after the intervention and with adjustment over baseline values.

Pearson correlation analysis was used to examine the relationship between the variables. Percentage change was calculated in each group using the difference percentage before and after the intervention. Data analysis was done using SPSS17 software and MsExcel2007 and at significance level of 0.05.

Results

Table 2 presents the Changes of physiological variables during study period.

After 8 weeks the results of repeated measure analysis performed in separate groups represented significant decrease over time in aerobic exercise group on fasting glucose variable ($F(2,18) = 8.9$, $P = 0.002$), but these changes were not significant for the control group ($F(2,18) = 1.44$, $P = 0.053$). Also the results of post hoc tests revealed that variations in the two aerobic exercise groups were due to the time difference; before the intervention and 4 weeks ($P= 0.008$) and 8 weeks after theintervention ($P = 0.010$). The results of analysis of covariance (ANCOVA) in comparison of mean value of fasting glucose variable between the groups after the intervention, and by adjusting the baseline quantities of these variables, no significant differences between experimental and control groups was detected after 4 weeks ($F(1,17) = 8.13$, $P = 0.011$) and 8 weeks ($F(1,17) = 20.24$, $P = 0.003$).

After 8 weeks of exercise analysis of repeated measurements performed separately in aerobic exercise group ($F(2, 18) = 1.69$, $P = 0.213$) and the control group ($F(2,18) = 2.27$, $P = 0.132$) the change in insulin variable was not significant.

The results of analysis of covariance (ANCOVA) in comparison of mean value of insulin variable between the groups after the intervention, and by adjusting the baseline quantities of these variables, no significant differences between experimental and control groups was detected after 4 weeks ($F(1,17) = 0.18$, $P = 0.679$) and 8 weeks ($F(1,17) = 0.16$, $P = 0.692$).

After 8 weeks of exercise analysis of repeated measurements performed separately in aerobic exercise group ($F(2, 18) = 1.84$, $P = 0.188$) and the control group ($F(2, 18) = 2.32$, $P = 0.159$) the change in insulin resistance variable was not significant.

Table 2. Changes of physiological variables during study period. Data are expressed as mean ± standard deviation.

Variables	Arobic training group			Control group		
	Pre test	Mid-test	Post-test	Pre test	Mid-test	Post-test
VO2max (mm/kg/min)	25.77 ± 2.32	29.78 ± 2.26	32.2 1± 1.54	25.81 ± 2.18	26.54 ± 2.46	27.02 ± 2.49
Body fat (%)	9.46 ± 0.83	9.64 ± 0.79	9.42 ± 0.72	10.05 ± 1.08	10.22 ± 1.01	10.22 ± 0.83
BMI (kg/m²)	27.16 ± 1.46	26.81 ± 1.48	26.56 ± 1.35	27.86 ± 2.03	28.23 ± 1.80	28.11 ± 1.69
Weight (kg)	70.60 ± 6.04	69.70 ± 6.02	69.00 ± 5.33	69.30 ± 7.92	70.30 ± 8.39	70.00 ± 8.30

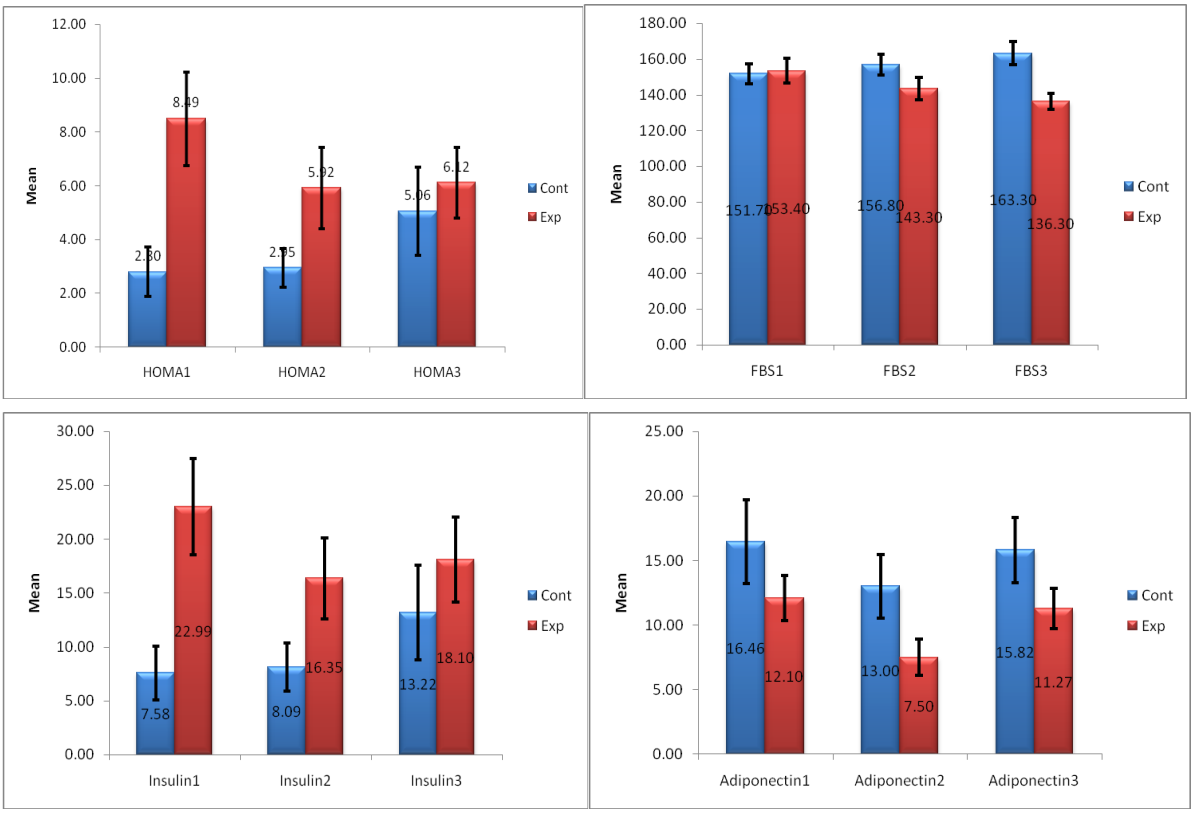

Fig. 1. Concentrations of fasting glucose (mg/dl), insulin (U/ml), insulin resistance (HOMA) and Adiponectin (ng/ml) at Pre, Mid and Post test in Aerobic and control groups.* Denotes statistical differences from Control group. Values are mean ± SD.

The results of analysis of covariance (ANCOVA) in comparison of mean value of insulin resistance variable between the groups after the intervention, and by adjusting the baseline quantities of these variables, no significant differences between experimental and control groups was detected after 4 weeks (F (1,17) = 0.17, P = 0.689) and 8 weeks (F (1,17) = 0.14, P = 0.710).

After 8 weeks the results of repeated measure analysis performed in separate groups represented significant decrease over time in aerobic exercise group on adiponectin variable (F (2, 18) = 16.03, P = 0.000, but these changes were not significant for the control group F (2, 18) = 6.39, P = 0.008). Also the results of post hoc tests revealed that variations in the two aerobic exercise groups were due to the time difference; before the intervention and 4 weeks (P = 0.006) and 8 weeks after the intervention (P = 0.004)

and for control group the differences were respectively between before intervention and 4 weeks (P = 0.001) and 8 weeks (P = .001) after the intervention.

The results of analysis of covariance (ANCOVA) in comparison of mean value of adiponectin variable between the groups after the intervention, and by adjusting the baseline quantities of these variables, no significant differences between experimental and control groups was detected after 4 weeks (F (1,17) = 4.97, P = 0.040) and 8 weeks ((F (1,17) = 1.10, P = 0.310); there was a significant decrease.

After 8 weeks the results of repeated measure analysis performed in separate groups represented significant decrease over time in aerobic exercise group on weight variable F (2,18) = 4.49, P = 0.026), but these changes were not significant for the control group (F (2,18) = 1.65, P = 0.220). Also the results of post hoc tests revealed that variations in the two aerobic exercise groups were due to the time difference; before the intervention and 8 weeks after the intervention (P = .041).

The results of analysis of covariance (ANCOVA) in comparison of mean value of insulin resistance variable between the groups after the intervention, and by adjusting the baseline quantities of these variables, no significant differences between experimental and control groups was detected after 4 weeks (F (1,17) = 6.60, P = 0.020) and 8 weeks (F (1,17) = 4.87, P = 0.041).

After 8 weeks the results of repeated measure analysis performed in separate groups represented significant decrease over time in aerobic exercise group on BMI variable F (2,18) = 4.49, P = 0.026), but these changes were not significant for the control group (F (2,18) = 1.65, P = 0.220). Also the results of post hoc tests revealed that variations in the two aerobic exercise groups were due to the time difference; before the intervention and 8 weeks after the intervention (P = .041).

The results of analysis of covariance (ANCOVA) in comparison of mean value of BMI variable between the groups after the intervention, and by adjusting the baseline quantities of these variables, no significant differences between experimental and control groups was detected after 4 weeks (F (1,17) = 9.52, P = 0.007) and 8 weeks (F (1,17) = 9.15, P = 0.008).

After 8 weeks the results of repeated measure analysis performed in separate groups represented significant decrease over time in aerobic exercise group on BF% variable (F (2,18) = 1.69, P = 0.213), but these changes were not significant for the control group (F (2,18) = 2.27, P = 0.132).

The results of analysis of covariance (ANCOVA) in comparison of mean value of BF% variable between the groups after the intervention, and by adjusting the baseline quantities of these variables, no significant differences between experimental and control groups was detected after 4 weeks (F (1,17) = 0.18, P = 0.679) and 8 weeks (F (1,17) = 0.16, P = 0.692).

After 8 weeks the results of repeated measure analysis performed in separate groups represented significant increase over time in aerobic exercise group on VO2max variable (F(2,18)=4.86, P=0.020), and in the control group (F (2,18) = 71.75, P = 0.000). Also the results of post hoc tests revealed that variations in the two aerobic exercise groups due to differences between each pair of measurements that is; before the intervention (P=.000), 4 weeks after (P=.000); and 8 weeks after the intervention (P = .001). And in control group due to difference between the time before and after; before the intervention (P=.000), 4 weeks after (P=.049); and 8 weeks after the intervention (P = .011).

The results of analysis of covariance (ANCOVA) in comparison of mean value of VO2max variable between the groups after the intervention, and by adjusting the baseline quantities of these variables, there was a significant difference between

experimental and control groups was detected after 4 weeks (F(1,17)=33.41, P=0.000) and 8 weeks (F(1,17)=61.82, P=0.000).

Discussion and conclusion

The first result of this study shows that eight weeks of aerobic exercises would have no significant effect on the concentration of plasma adiponectin in women with type 2 diabetes. Unlike other adipocytokine, adiponectin levels are reduced in obese diabetics (Weyer *et al.,* 2001; Yu *et al.,* 2006). Another study reveals that diabetic and non-diabetic subgroups represent a greater difference in adiponectin concentrations in relation to body mass index (BMI) (Stejskal *et al.,* 2003), and this finding may be attributed to the fact that diabetes mellitus is more associated with decreased production of adiponectin than with obesity. However, some studies suggest that changes in blood concentrations of adiponectin is inversely related to body fat mass and its positive changes occur in association with weight loss and decreased muscle mass (Matsuzawa *et al.,* 2004; Chan *et al.,* 2005; Hisayo *et al.,* 2004). Studies in Japan show that plasma levels of adiponectin correlate inversely and significantly with BMI and plasma levels of adiponectin in obese individuals is lower than in lean individuals (Hotta *et al.,* 2000). Reduction in blood levels of adiponectin with increasing degrees of obesity can probably be due to decreased half-life of adiponectin molecules present in circulating blood in obese patients, which is caused by increased degradation of these molecules (Hoffested *et al.,* 2004); another possibility, however, is decreased metabolic function of adipocytes in tandem with their hypertrophy or aging (Hu *et al.,* 1996). There are two maturity phases in adipocytes: differentiation and hypertrophy. During the early stage of maturation (differentiation) adipocytes possess high levels of metabolic activity and fuel consumption increases. The young cells are relatively small and sensitive to insulin and show increased adiponectin. In contrast, the older cells grow in size (hypertrophy) and lose most of their functional activity which is adiponectin synthesis (Kern *et al.,* 2003).

Recent studies show that mitochondrial biogenesis increases during differentiation of adipocytes, but the number of mitochondria in adipocytes of obese mice db/db reduces. These data suggest that mitochondrial biogenesis may drastically require differentiation of adipose (metabolic activity) adipocytes hypertrophy and reduce the number of mitochondria (Eun *et al.,* 2007).

Mitochondrial function is essential for the differentiation of adipocytes and will shrink through increased size of mitochondria, differentiated cells' size, due to increased fatty acid oxidation and decrease intercellular triglyceride accumulation. The researchers found that levels of adiponectin synthesis would increase in small adipocytes, (Kern *et al.,* 2003) which are caused by an increase in mitochondrial function. In contrast, in hypertrophied fat cell, mitochondrial dysfunction declines due to adiponectin synthesis. In the present study the BF% not having reduced significantly despite significant reductions in weight and BMI can signify fat cell hypertrophy leading to non-significant results in adiponectin and significant weight and BMI loss may have been caused by factors other than the reduction of body fat percentage (such as decreased body water and protein, etc.) The results of research are consistent with the findings of Hisayo 2004, Kobayashi 2006 and Kon Ilim 2007 (Hisayo *et al.,* 2004; Kobayashi *et al.,* 2006; Kang *et al.,* 2007). BF% changes remaining insignificant in this study may result from failure to observe Glycemic and lipidemic index of foods eaten by the subjects due to not having the same diet. If subjects follow a diet containing foods with a high glycemic or lipidemic index, they will gain weight and because obesity is associated with mitochondrial dysfunction repeats the vicious cycle and poor mitochondrial function prevent increased synthesis of adiponectin. In this study the plasma adiponectin concentrations did not change significantly; consequently, lipid biosynthesis did not

decrease which is further supported by the subjects' subcutaneous fat not changing significantly. The results of our study, however, are contrary to the findings of Nemeth et al 2003, Dymetro et al 2006 and Zang et al (Nemet *et al.,* 2003; Dimitrou *et al.,* 2006; Zeng *et al.,* 2007), which suggest considerable impact of aerobic exercise on the increase of plasma adiponectin levels. (Kraemer *et al.,* 2007) In another study Kramer et al 2007 review the studies that have examined the effect of exercise on adiponectin concentrations and propounded this assumption that the volume of training seems to an influential factor in adiponectin response as long-term exercise of high volume (intensity, duration and frequency) influences the concentration of adiponectin. In this study too although plasma adiponectin level is insignificant, and as the plasma adiponectin level increases considerably yet insignificantly in aerobic exercise group in the fourth week compared to the first week, it is likely that increasing the length of each training session as well as the duration of the study in the second four weeks compared with the first four weeks have influenced these changes.

Furthermore, mitochondria as one of the major organelles producing ROS oxidative stress when suffering performance degradation caused by obesity, may increase production of ROS and reduce adiponectin synthesis (Chevillotte *et al.,* 2007). So, probably due to insignificant effect aerobic exercises on significant reduction of BF%, hypertrophied fat cells would cause poor mitochondrial function and increased production of ROS not having caused any significant changes in plasma adiponectin levels.

In addition, studies suggest that spherical and chain adiponectin stimulates phosphorylation and activity of AMPK in the skeletal muscles. Aligned with the activity of AMPK, adiponectin, phosphorylation and stimulation of acetyl coenzyme causes A Carboxylase (ACC) and consequently stimulation of ACC inhibits and decreases Malonyl-CoA which inhibits intake of fatty acids consumed in the mitochondria and further increases oxidation of fatty acids and decreases biosynthesis of fat (Kadowaki *et al.,* 2006; Kershaw *et al.,* 2004; Barnevholm *et al.,* 2003). In addition, adiponectin indirectly stimulates lipoprotein lipase (Coms et al., 2004) and since the impact of adiponectin on cells increases consumption of fatty acids and facilitates entry of sugar into the cell, this impact increases sensitivity of cells to insulin (Berggren *et al.,* 2005; Behre, 2007). On the other hand injection of adiponectin to the body improves insulin sensitivity of cells in obese diabetic subjects (Matsuzawa *et al.,* 2004). As mentioned mitochondrial function in in skeletal muscle is in relation to insulin sensitivity of the entire body; therefore the synthesis of adiponectin is regulated by mitochondrial function in adipocytes. Thus, it is likely that mitochondrial activity of adipocytes regulates insulin's function in skeletal muscle through adiponectin. Mitochondrial dysfunction is a separate determinant of insulin resistance in different tissues of obese individuals (Eun *et al.,* 2007).

Also Yamvochyi et al 2001 (Yamauchi *et al.,* 2001) showed in a study that obesity by induced high-fat diet would decrease the expression and blood levels of adiponectin, leading to insulin resistance. Adiponectin influences the intracellular function, since it has been observed that the decrease in tyrosine phosphorylation of insulin receptors of muscular cells is associated with low concentrations of plasma adiponectin which is symptoms of the onset of diabetes.

These findings are very valuable clinically because IRS-1 tyrosine phosphorylation and PI3K activity decreases in skeletal muscle in type II diabetic patients (Matsuzawa *et al.,* 2004). On the other hand adiponectin increases the mitochondrial number and function in skeletal muscle by protein kinase activated by 5-AMP and mitochondrial function skeletal muscle is noticeable as an important determinant of whole-body insulin sensitivity (Eun *et al.,* 2007). Thus if exercise is effective in mitochondrial function and consequently on the increase of plasma adiponectin, it will improve insulin

sensitivity and is important not only as a kind of therapy but also as an appropriate and economic approach in preventing type II diabetes (Stefan *et al.,* 2002).

In this context Belhor et al (Bluher *et al.,* 2004) also report significant blood increase of blood levels of adiponectin after four weeks of aerobic exercise caused by increased insulin sensitivity. But ultimately, the second finding of our study that is the eight-week aerobic exercise not affecting insulin resistance is not consistent with such results and this inconsistency may be due to the lack of control of hormonal factors such as glucocorticoids, thyroid hormones, growth hormone and angiotensin II which disrupt glucose tolerance or insulin resistance or the volume (duration and intensity) of the exercise may not have been adequate to cause changes while the statistical results of the research becoming insignificant may be due to the small sample size, which reduces the statistical power. On the whole; the results of this study suggest that 8 weeks of aerobic exercise would not have any significant impact on plasma adiponectin and insulin resistance in women with type II diabetes. It, however, has a significant effect on weight, BMI and fasting glucose in this group of diabetic women. In addition, 8 weeks of aerobic exercise significantly influenced VO2max. Therefore, on the basis of results obtained from this the study it is recommended that patients with type 2 diabetes capable of performing aerobic exercise should benefit from the useful effects of this type of exercise as a method for controlling glycemic variables.

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