

CLINICAL STUDY

The effects of three training methods endurance, resistance and concurrent on adiponectin resting levels in overweighted untrained men

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Abstract: The purpose of present study was to investigate the impacts of endurance, resistance and concurrent training on adiponectin resting levels of sedentary men. Forty-four sedentary students were randomly assigned to one of four groups: endurance training (ET; 22 ± 0.89 yr, $n=12$), resistance training (RT, 21 ± 1.57 yr, $n=9$), concurrent training (CT, 21.38 ± 2.6 yr, $n=14$) and control group (CG, $n=10$). After primary measurements, blood samples were drawn with subjects in fasting and resting state for determination of the basic level of adiponectin. The subjects participated in E, R and C training for 8 weeks. The ET group ran 3d/w at 65–85 % of maximum heart rate. The exercise training session for the RT group consisted of 3 sets of 10–15 repetitions of weight training exercise that increase progressively, and repeated 3 sessions per week. The CT group trained exactly the sum of ET and RT groups. Correlated samples t-test and ANOVA were used. The results of the present study showed that after the eight-week training, the adiponectin levels of subjects increased in 3 groups of training but this increase was not significant. The level of adiponectin in CT group increased more than in ET and RT groups. Also, there were no significant differences in content of adiponectin among groups. In general, slight increases in adiponectin levels in training groups especially in CT group may indicate the most potential of CT group in increasing the levels of adiponectin in sedentary men. However more researches are needed to identify the effects of concurrent training

(Tab. 4, Ref. 27). Full Text in PDF www.elis.sk.

Key words: adiponectin, endurance training, resistance training, concurrent training.

Adipose tissue is a source of many factors derived from adipocyte. Adipocytokines point to active biologic molecules that are derived from fat cells. Some of these adipocytokines mediate the systemic effect of obesity. Recently, adiponectins have been recognized as adipocytokines with an important metabolic effect (11, 12).

Adiponectin is a protein with 244 amino acids and is released in a large amount from human adipose tissue. The contrast of this cytokine is high in blood, and in fact it contains 0.01 percent of the total of serum proteins (17). Although adiponectin is released only from white adipose tissue, unlike most adipokines its gene expression in white adipose tissue and serum contrasts the decrease in obesity and overweight. Beside its antiinflammatory effects, the important effect of adiponectin that has been taken into consideration is its relationship to risk factors of metabolic syndrome.

The results of some researchers have demonstrated that a decrease in adiponectin relates to insulin resistance, hyperinsulinism and hyperglycemia (21).

Furthermore researches on different races have reported that adiponectin levels increase after weight loss and improvement of insulin resistance (5, 20).

When compared to other adipocytokines, plasma adiponectin decrease after obesity and insulin resistance. It is described that maybe other adipocytokines have their effects on insulin resistance by affecting adiponectin gene expression and its release (13). Adiponectin may affect fatty acid oxidation in skeletal muscle and consequently decrease TG in skeletal muscle fibers and increase lipoproteins catabolism. Nevertheless adiponectin may decrease the flow of fatty acids to the liver and production of liver glucose (13).

Also it may be that adiponectin has antiatherogenic and anti-inflammatory effects while it acts as a mediator between obesity and atherosclerosis (18).

One of assumed mechanisms of how exercise can decrease inflammatory processes is via weight loss. Weight loss can decrease cytokines production from adipose tissue (7). Of course one of effective factors in altering the body composition especially that of adipose tissue is the balance between energy intake and consumption. To change the body composition, it is the endurance training that is usually used due to its ability to increase energy expenditure

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and use fats. Although the results of previous studies investigating the effects of endurance training on BMR are different, some of them show an increase in RMR (1, 3, 24) but other show no difference in BMR (19) or a decrease in RMR (3).

It is reported that different factors affect the amount of metabolism. The strongest relationship is between FFM and BMR. It is thought that increasing the lean body mass increases BMR and then total energy expenditure increases (15).

Generally, fat mass and total body mass decrease with endurance training, however this decrease interferes very little with the increase in lean body mass (23). Many researches focus on increasing lean body mass with resistance training. Although some researches have been done on the effect of training on adiponectin concentrations and its relation with increasing insulin sensitivity, in most of them it is reported that plasma adiponectin concentrations have not changed with training in healthy subjects. For example Hulver et al (2002) reported insulin activity improved after six-month endurance training but plasma adiponectin concentrations did not change in healthy subjects (6). Plasma adiponectin did not increase in healthy young men after a long walking program (10). Also in elite rowers, plasma adiponectin did not increase significantly in training season (4), but after a six-month endurance training plasma adiponectin concentrations decreased by 18 % compared with pre-training in healthy men (25). Zeng et al (2007) demonstrated that exercise-induced changing in total plasma adiponectin depends on intensity and duration of training (26). Few studies have been done on resistance training. Some of them demonstrated that resistance training significantly decreased the insulin resistance index in obese men but it did not affect serum adiponectin significantly.

Hara et al (2005) used aerobic and resistance training with each other and reported no difference in adiponectin (4). Concurrent training is a recently new method in training programs. Researchers demonstrated positive effects on body composition by concurrent training such as increasing FFM, and decreasing FM and body fat percentage. In another way Jurimae et al (2005) hypothesized that if energy consumption is intensified during exercise and has higher metabolic pressure, the probability of increasing the exercise-induced adiponectin is greater (8). So, by paying attention to few researches about the effect of concurrent training on adiponectin and its effect on energy expenditure and body composition especially fat mass and fat percentage, it is required to study the effect of such a training program and compare these two traditional methods (endurance and resistance) on adiponectin changes. As a result the aim of present study was to demonstrate the effect of three training methods, namely endurance, resistance and concurrent on adiponectin in sedentary healthy men.

Methods

Subjects

Forty-four sedentary healthy college students took part in this study (Tab. 1). They did not have former regular exercise training and while being awarded with the goal of the study they signed a letter of satisfaction. Then they were divided to four groups:

resistance training (9 persons), endurance training (12 persons), concurrent training (13 persons) and control group (10 persons). At first height, weight, fat percentage, BMI and $VO_2\max$ of subjects were evaluated (Tab. 1).

Training protocol: After primary evaluations and measuring the basic levels of indexes, the subjects took part in resistance, endurance or concurrent trainings three days per week for 8 weeks.

Enduring training program: Endurance training program was composed of a running program with duration and intensity increased gradually at the end of each level. In first and second weeks, the subjects worked out at 65 % of MHR for 25 minutes. From third until the sixth week they worked out at 65–75 % of MHR for 35 minutes and from seventh till eighth week at 75–85 % of MHR for 40 minutes. For controlling their heart rate, polar belts were used.

Resistance training group: The subjects of this group took part in a resistance training program with machines and free loads. The training program contained upper body training and lower body training such as bench press, sitting & standing up with halter, leg extension, leg flexion, leg press and rowing.

In the first two weeks, subjects did three sets with 10–15 repetition and in the next six weeks their training was set for them in a way that they could not do 10–12 repetition in first set, 8–10 repetition in second set and 4–8 repetitions in third set.

Concurrent training group: These subjects took part in a training program that exactly summed the endurance and resistance training. For this group endurance and resistance training programs were done exactly in the same days with two other groups but the resistance training was always done first.

Adiponectin measuring

For measuring adiponectin, blood samples were taken after 12-h fasting, and after separating the serum it was stored at -80°C . Adiponectin measuring was done with ELISA and its special kit (Biovendore).

Statistic methods

Descriptive statistics was used to assign mean and standard deviations. Kolmagrov-Smirnof was used for defining normal distribution. Change in each group was demonstrated with dependent T while for comparing changes between groups ANOVA was used.

Results

Table 1 shows mean and standard deviations of age, height, weight, body mass and $VO_2\max$.

Table 2 shows mean and standard deviations for adiponectin before and after training. The results of adiponectin levels after training indicate that in concurrent group they are highest among all groups.

Table 3 shows adiponectin concentrations before and after training in each of the training groups and in control group.

The results of Table 2 show that there is no significant difference between adiponectin concentrations before and after training in endurance, resistance and concurrent training groups.

Tab. 1. Mean and standard deviations of age, height, weight, body mass and VO₂max.

Groups	Endurance (n=12)		Resistance (n=9)		Concurrent (n=13)		Control (n=10)	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
BMI	29.86±3.94	28.70±3.59	31.48±4.95	30.44±4.80	28.64±3.76	27.38±3.67	29.26±4.27	29.29±4.40
VO ₂ max	27.72±5.99	33.75±5.38	26.10±4.47	32.49±4.04	27.83±3.76	35.64±6.44	31.19±6.39	31.02±5.00
weight	90.55±10.80	87.50±11.07	92.22±15.85	89.17±15.29	86.61±15.05	83.46±14.02	90.00±14.05	90.33±14.60
height	174.64±4.72	--	171.11±5.51	--	173.65±7.07	--	175.61±5.09	--
age	22.0±0.89	--	21.0±1.57	--	21.38±2.06	--	21.44±1.13	--

Tab. 2. Adiponectin mean standard deviations before and after training in each group.

	Group	Amount	Mean	SD
Adiponectin before training	Endurance	12	16.67	2.35
	Resistance	9	16.67	2.35
	concurrent	13	17	5.37
	control	10	20.30	8.35
Adiponectin after training	Endurance	12	17.56	1.51
	Resistance	9	17.56	1.51
	concurrent	13	20.38	7.61
	control	10	18.80	2.69

The result of ANOVA is shown in Table 4 and indicates that adiponectin had no significant difference between the groups.

The results of present study demonstrate that although there was a little increase in all three groups but there is no significant difference in adiponectin concentration before and after training in three groups. And also there is no significant difference in adiponectin concentrations between the groups.

Conclusion

Although the results of present study show increasing values after training in all groups they were not significantly meaningful within groups as well as between groups. Naturally the increase in adiponectin was the greatest in the concurrent group. The resulting changes achieved in this study on adiponectin are similarly insignificant as those achieved by Yatagai (2003), Kobayashi (2006), Rouzen Bavem (2007) and Jurimae (2006).

Yatagai et al (2003) studied adiponectin concentrations after six-week rowing on ergometer (five sessions per week at the intensity of lactate threshold) in healthy men with normal weight. The results show that BMI and fat mass did not change after six weeks of training. The amount of adiponectin hormone decreased 16 hours after training but it came back to normal concentrations after a week. Researchers concluded that in healthy persons, increasing exercise-induced insulin sensitivity has no relation to increasing of plasma adiponectin and it seems that decreasing the fat mass and BMI of subjects after training have a role in increasing the adiponectin concentrations (25).

Tab. 3. Adiponectin concentrations before and after training.

	Mean ± SD		Amount	df	t	p	Result
	pre	post					
Endurance	16.67±2.35	17.56±1.51	12	11	-1.502	0.164	Ns
Resistance	16.67±2.35	17.56±1.51	9	11	-0.951	0.369	Ns
Concurrent	17.00±5.37	20.38±7.61	13	12	-1.087	0.298	Ns
Control	20.30±8.35	18.80±2.69	10	9	0.513	0.621	Ns

Tab. 4. The results of variance for comparing changes between groups.

	df	F	P
Between group	3	0.768	0.519
Within group	40		
Total	43		

Kobayashi et al (2006) studied the effect of a walking program (50 days with a mean of 8211±2084 steps/day) on metabolic parameters such as adiponectin in healthy men. Their results show a significant decrease in BMI, TG, TNF_α, after their training program, nevertheless plasma adiponectin, fat weight and fat percentage did not change. By paying attention to increasing the adiponectin following a significant decrease in body weight, the researchers reported that the probable reason for invariability of adiponectin in their study was the intensity of training program that did not decrease the weight and body fat percentage (10). In another study that was done by Rouzen Bavem (2007), 73 students of eighth grade took part in aerobic training program (rhythmic performance, 3 sessions/week) for 3–4 months. At the end of the program, the body fat percentage and insulin sensitivity decreased but adiponectin concentrations did not change (16).

Jurimae et al (2006) studied the plasma adiponectin response to training in elite rowers in training season. They took part in specific training for 6 months. Plasma adiponectin was measured before and after the test. The results show that after six months plasma adiponectin changed in neither of groups in resting state (8).

By paying attention to results of previous studies it seems that in healthy persons with normal weight, plasma adiponectin concentration is not affected by long-term training program. In another way Polak et al (2006) studied the effect of aerobic training program on plasma concentration of adiponectin and TNF_α, and both adiponectins mRNA. The subjects of that study were 25 sedentary premenopause obese women (BMI= 32.2±2.2 kg/m²). They were subduced to aerobic training for 45 minutes each day, 5 days/week for 12 weeks with 50 % VO₂max.

The results show that their weight and fat mass decreased by 5.9 % and 6.4 %, respectively. Also there were no changes in plasma adiponectin and plasma TNF_α and the expression of both adiponectins in adipose tissue. The researchers described two hy-

potheses for this invariance. Firstly, the aerobic training in this research did not lead to any significant weight loss and thus did not affect adiponectin. Secondly, it seems that exercise-induced increasing of catecholamines can inhibit adiponectin gene expression and its amount in plasma (18). So by paying attention to invariance of subject's weight in this research, these mechanisms can interfere with adiponectin changes (18).

Kelly et al (2007) studied the effect of training on plasma adiponectin and TNF_{α} in children with overweight (BMI>30). The subjects were 20 children at age of 10–12 divided in two groups, namely training group and control group. The training program contained four ergometer sessions/week for eight weeks. The intensity in the first week was at 50–60 % of VO_2 max and in the eighth week it was at 70–80 % of VO_2 max. The duration of each session in the first week was 30 minutes and gradually increased up to 50 minutes in the eighth week. The results show that weight, BMI, body fat percentage and upper body fat percentage did not significantly change. Also TNF_{α} and adiponectin did not change after training. The researchers concluded that exercise that neither decreased weight nor improved body composition, could not affect adiponectin, and if so, only with little effect (9).

As mentioned, in three previous studies the researchers concluded that the reason of invariability of adiponectin concentration after training in obese persons or in persons with overweight lied in the fact that weight or body composition did not decrease or improve respectively. However, Allery et al (2005) reported that after an aerobic training program, the body composition and especially abdomen visceral fat improved but plasma adiponectin did not change. These researchers studied the effect of a twelve-week aerobic training on glucose metabolism, body composition and plasma adiponectin in 16 obese old men and women (at mean age of 61 years, BMI=33.2±1.4 kg/m²) who took part in an aerobic exercise (running) on a treadmill or ergometer five days/week for 60 minutes at 85 % of MHR). The results show that exercise improved insulin resistance and decreased the body weight, fat mass, visceral and subcutaneous fat and abdominal visceral fat, nevertheless plasma adiponectin and TNF_{α} did not change. Also there was not any correlation between adiponectin and abdominal visceral fat changes (14).

Hara et al (2005) studied the effect of aerobic training and concurrent training (aerobic and resistance) on plasma adiponectin hormone. The subjects of their study were young obese men (BMI=31.1±4.2 kg/m²) who were divided in three groups (aerobic, concurrent and control). Aerobic training program was composed of eight weeks of running on a treadmill or ergometer in three sessions/week while each session lasted more than 30 minutes at 54.8 % of VO_2 max. In the concurrent group the aerobic training was done plus resistance training for five months. Body composition and adiponectin have been measured before and after training. In aerobic training group, the body weight and BMI did not change whereas fat mass decreased significantly. Also adiponectin increased by 8.1% in this group but it was not significant. In concurrent group, the body weight, BMI, fat percentage and fat weight decreased significantly and adiponectin increased by 6.5 % but it was not significant statistically. It is reported that the amount of

changes in adiponectin have significant negative correlation with the decrease in body weight, BMI and fat mass. The results of regression analysis also distinguished the body fat percentage to be an anticipator of plasma adiponectin. The researchers reported that it seems that exercise-induced changes in adiponectin took place via changes in body composition, so for increasing plasma adiponectin in obese men, a training that improves body composition is required (4). There are different studies about the effect of exercise on plasma adiponectin in subjects with insulin resistance and diabetes II. In some of them it is reported that plasma adiponectin or its mRNA in adipose tissue increased after exercise (2) but in others there were no significant changes. In people that had resistance training records or they were runners and also did resistance training, adiponectin levels increased by 30% and 37 %, respectively after a session of weight training (22). Generally it seems there are different reasons and mechanisms in invariability of adiponectin in present study and that one of them may lie in the unchanged body weight of subjects. Also the duration and intensity of training are important. A small increase in adiponectin in three groups especially in the concurrent group shows that this program has a greatest effect on changing adiponectin levels in untrained people. It seems that more studies with longer duration are required for distinguishing the exact effects.

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