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Anti-inflammatory effect of aerobic program in obese men

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Key words: Exercise, obesity, TNF-α, loss weight.

Abstract

The aim of this study was to evaluate the effects of exercise training on serum TNF-α levels obese men. For this purpose, twenty-eight obese sedentary males (age, 41 ± 5 years) were divided to exercise and control groups. Exercise groups underwent a 12-week aerobic exercise program, with a frequency of 3 d/wk and intensity corresponding to 60-80% of individual maximal heart rate and the control subjects were instructed to maintain their habitual activities. Bodyweight and other anthropometrical markers and serum TNF-α were measured before and after the 12 week intervention. Compared to pre-training, the TNF-α level decreased significantly after exercise program in the exercise, but not in the control groups. With aerobic exercise training, subjects in exercise group lost bodyweight, BMI and body fat percentage ($p<0.05$). Based on these findings, we can say aerobic exercise is associated with low inflammation cytokine in obese men even.

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Introduction

According to the population studies, obesity is associated with low grade inflammation and inflammation cytokines are produced depend on adipose tissue level in obese subjects (Moschen *et al.,* 2010). A large body of evidence suggests that adipose tissue produced a variety of bioactive mediators including adipocytokines such as leptin and adiponectin or classical cytokines such as the pro-inflammatory mediators tumor necrosis factor a (TNF-α) (Hotamisligil, 2006; Tilg *et al.,* 2006).

Plasma concentrations of have been positively correlated with increased triglycerides (Kern *et al.,* 1995). It was established that this inflammation cytokine is produced 7.5 times more by the adipose tissue in obese subjects compared with lean counterparts (Kern *et al.,* 1995). It has been established that expression of TNF-α, a major pro-inflammatory cytokine, increased in fat cells from obese subjects and those with insulin resistance and is markedly regulated at the transcriptional level (Rotter *et al.,* 2003).

Recent studies have shown that TNF-α is play a critical role in the pathogenic mechanisms of a number of chronic inflammatory diseases (Cristina *et al.,* 2005). Binding of this cytokine to its receptors results in activation of intracellular signaling processes, which lead to release of pro-inflammatory mediators and apoptosis (Wajant *et al.,* 2001). It was reported that weight reduction is associated with decreased serum TNF-α (Dandona *et al.,* 1998).

The aim of the present study was to investigation the effects of a 3-month aerobic exercise training program in on serum TNF-α and anthropometrical indexes in sedentary adult obese men. We hypothesized that exercise would lead to an improvement in this inflammation cytokine in these subjects.

Materials and methods

Subjects

Twenty-eight apparently healthy obese men were recruited for this study through local advertising. Subjects were 35–45 years old with a body mass index (BMI) of 30–36 kg/m2. Participants were divided into exercise and control groups by randomly. This study examined the effect of aerobic exercise program on serum TNF-α in studied subjects. Informed consent was obtained from each subject after full explanation of the purpose, nature and risk of all procedures used.

Inclusion and exclusion criteria

Participants were included if they had not been involved in regular physical activity/diet in the previous 6 months. A detailed history and physical examination of each subject was carried out. Subjects with any history of smoking, chronic cough, recurrent respiratory tract infection, history of chest or spinal deformity, diabetic, personal history of asthma, chronic obstructive lung diseases were excluded from the study. Participants were non-athletes, non-smokers and non-alcoholics. In addition, exclusion criteria included inability to exercise and supplementations that alter carbohydrate-fat metabolism.

Measurements

Anthropometrical and blood samples were measured before and after exercise program. All measurements were carried out in the morning during the post absorptive phase. Weight and height of the participants were measured by the same person when the participant had thin clothes on and was wearing no shoes by using the standard hospital scales. Body composition monitor (BF508-Omron made in Finland) with a precision error of less than 100 g was used to measure weight and body fat percentage of the subjects. Body Mass index (BMI) was calculated using the formula body weight/height2 in terms of kg/m². Abdominal circumference was measured in the most condensed part using a non-elastic cloth meter. Blood samples

were collected after an overnight fast (10-12 hours fasting) before and 48 hours after last session of exercise program in order to measuring serum TNF-α. All measurements were done before and after this period in control group. Serums were immediately separated and stored at -80° until the assays were performed. Serum TNF-α was determined by ELISA method (Enzyme-linked Immunosorbent Assay for quantitative detection of human TNF-α total). The Intra- assay coefficient of variation and sensitivity of the method were 7.7% and 5.0 pg/mL, respectively.

Table 1. Mean and standard deviation of anthropometrical and biochemical variables of studied groups in baseline and after intervention.

Group	Experimental group		Control group	
Variable	baseline	post-test	baseline	post-test
Age (year)	42 ± 6.3	42 ± 6.3	41 ± 5	41 ± 5
Weight (kg)	101.2 ± 7.9	100.8 ± 8.9	100 ± 6	93 ± 6.5
Height (cm)	174.1 ± 7.8	174.1 ± 7.8	173.5 ± 6	173.5 ± 6
Waist circumference (cm)	107 ± 8.8	108 ± 9.1	106.3 ± 7.5	98 ± 7.8
BMI (kg/m2)	33.38 ± 3.11	33.25 ± 3.21	33.22 ± 2.78	30.89 ± 2.73
Body fat (%)	32.18 ± 4.25	32.56 ± 4.95	31.86 ± 3.45	26.78 ± 4.16
TNF-α (	39.2 ± 7.3	37.9 ± 6.8	37.5 ± 6.5	27.5 ± 5.9

Exercise program

Subjects trained under supervision three times (60 min) per week for 12 wk at intensity of 60-80% of HRmax. Subjects received feedback if training intensities were either too high or low in comparison with desirable intensities. After a warm-up, subjects trained for approximately 30 - 45 min and 5–10 min of cool down activity. Aerobic exercises in each session included walking on a treadmill and stationary cycling. Subjects were contacted if an exercise session was missed. In this 12-week period, participants in the control group were barred from participating in any exercise training.

Statistical analysis

Statistical analysis was done for all the parameters. Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. Independent t-test was used to compare the means of variables between asthma and non-asthma groups. Student's t-tests for paired samples were performed to determine significance of changes in variables by exercise test in studied subjects. All statistical tests were performed and considered significant at a $P \leq 0.05$.

Results

In this study, we investigated the effect of a chronic aerobic exercise program for three months on serum TNF-α and anthropometrical indexes such as BMI and body fat percentage in sedentary adult obese men. Baseline and post training anthropometrical indexes and serum TNF-α of exercise and control groups are shown in Table 2. All values are given as mean and standard deviation. At baseline, serum TNF-α level as an inflammatory cytokines were the same in the exercise and control subjects ($p = 0.452$). No baseline differences were found between groups for any body weight, body mass index, body fat percentage and abdominal obesity (see table 1).

The data of paired T test showed that aerobic exercise program resulted in a significant decrease in body mass indexes in exercise group ($p = 0.021$), but this variable remained without change in control group ($p = 0.213$). Additionally, body fat percentage and abdominal circumference decreased significantly in response to aerobic training program in exercise group ($p \leq 0.05$) but not in control group ($p \geq 0.05$). Compared to pre-training, serum TNF-α decreased

significantly (p = 0.011) after exercise program but it was not changed in control group (p = 0.312, Fig. 1).

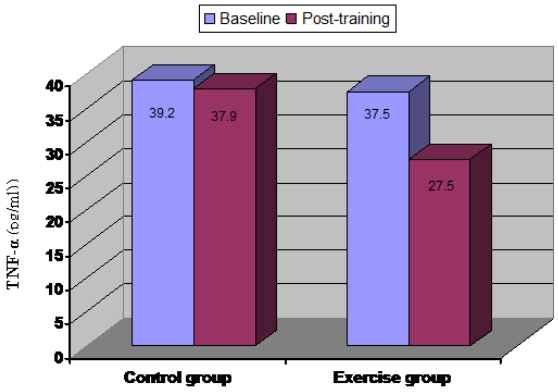

Fig. 1. The changes pattern of TNF-α in baseline and by interventions in two groups. The results showed that this inflammatory cytokine was decreased in response to three months of aerobic exercise when compared to baseline levels (p = 0.011).

Discussion

Our study data showed that three months aerobic training improves serum TNF-α as a pro-inflammatory cytokine in obese men. At present study, we also observed that aerobic training led to significant decrease in body weight, body mass index, and abdominal obesity in studied subjects. It seems that decreased serum TNF-α is created by weight reduction by exercise program. Obesity is known to be associated with a chronic inflammatory response characterized by abnormal cytokine production and activation of inflammatory signaling pathways (Moschen *et al.,* 2010).

Relation between obesity and a TNF-α as a pro-inflammatory cytokine came from a study in 1993, and the authors established the concept of a role for T TNF-α /inflammation in obesity (Hotamisligil *et al.,* 1993).

Patho-physiologically, it has been long known that cytokines act as catabolic factors involved in the pathogenesis of muscle wasting and cardiac cachexia (Anker *et al.,* 2004). There is considerable evidence that inflammatory cytokines have direct catabolic effects on skeletal muscle. For example, TNF-α impairs muscle protein synthesis (Lang *et al.,* 2002; Lang *et al.,* 2007) and increases muscle protein degradation (Li *et al.,* 2005; Tan *et al.,* 2006). On the other hand, increased serum TNF-alpha has been found in patients with reduced skeletal muscle cross-sectional area and peripheral muscle strength (Niebauer, 2000). The exact source of the circulating TNF-α is not fully understood, but this circulating cytokine is likely derived from the adipose Tissue depot and/or peripheral blood mononuclear cells (Charles *et al.,* 2008). It has been hypothesized that the increase in adipose tissue as one ages or the increase in secretion of cytokines from peripheral blood mononuclear cells could cause the loss of muscle mass (Visser *et al.,* 2002).

Increased TNF-α expression was observed in adipose tissue of obese subjects and reduced TNF-α serum levels are observed following weight loss (Cesari et al., 2005). There is evidence that weight reduction by exercise but not diet skeletal muscle inflammatory gene expression in frail obese elderly persons (Charles *et al.,* 2008). A recent study has established TNF-α mRNA and CD68+ cells were reduced by weight loss in adipose tissue where their abundance is high but there was no effect of weight loss on skeletal muscle TNF-α mRNA, and CD68+ cells where the abundance of CD68+ cells is low (Bruun *et al.,* 2006). On the other hand, it has been suggested that the lack of significant effect of weight loss on inflammatory markers (IL-6, and TNF-α mRNA) in skeletal muscle may be related to the fact that there appears to be a low abundance of macrophages (CD68+ cells) in skeletal muscle (Bruun *et al.,* 2006). Recent findings demonstrate that aerobic exercise is associated with low inflammation cytokine in obese men (Eizadi *et al.,* 2011). In present study, three months aerobic exercise program decreased serum TNF-α and body weight or body fat percentage. In fact, our study data indicates that exercise induced weight los is associated with decreased serum TNF-α in obese men. But, study by Mingrone and colleagues shoed that a 4.6% decrease in body weight did not significantly decrease TNF-α mRNA (Mingrone *et al.,*

2002). Based on these data, it seems that weight loss in above mentioned study was not enough to reduce inflammation cytokines. To support this hypothesis, the authors citing their findings suggest that a minimum weight loss of 5% is required to improve adipokine profile and decrease fat cell size in severely obese subjects (Varady *et al.,* 2009).

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