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Aerobic training program is associated with improved systemic inflammation in smokers

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Abstract

It was reported that smoker individuals are more prone to systemic inflammation than non-smokers. The objective of this study was to determine serum TNF-α response to a long term aerobic training in smoker men. For this purpose, thirty middle-aged healthy smokers selected for participate in this study and were divided into control and exercise groups by randomly. All subjects in exercise group were completed three months aerobic exercise training for 3 times weekly and the subjects of control group were banned on any regular training in this period. Anthropometrical and serum TNF-α were measured before and after this program in two groups. Student's t-tests for paired samples were performed to determine whether there were significant within-group changes in the outcomes. At baseline there were no differences in the anthropometrical and biochemical parameters between the two groups. Compared to pre-training, the TNF-α level decreased significantly after aerobic program in exercise group, but not in control group. Anthropometrics variables decreased significantly after exercise intervention in exercise groups. Based on these finding, we can say that regular exercise training is associated with improved systemic inflammation in smoker men

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Introduction

Some recent sources have introduced TNF-α as the main chemical mediator of inflammatory responses in the face of negative bacterial infections and other infectious germs and recognize it as being responsible for development of generalized complications in acute infections. Although adipose tissue is the major source of secretion of this inflammatory cytokine it is also discharged by macrophages and other cells. The positive and significant correlation of TNF-α with blood triglyceride levels, has been reported as a cardiovascular risk factor (Jovinge *et al.,* 1998). The stimulating role of TNF-α in the increased production vLDL has been observed in some studies which describes the relationship of this cytokine with TG Plasma (Qin *et al.,* 2008). It has also been noted that higher than normal levels of this inflammatory cytokine, inhibits muscle protein synthesis (Lang *et al.*, 2007; Lang *et al.,* 2002; Williamson *et al.,* 2005). The inhibitory effect of TNF-α inflammatory cytokine on the expression and serum levels of adiponectin has been reported in some literature (Puglisi *et al.,* 2008). Despite the available findings on the potential impact of inflammatory diseases on TNF-α, some studies support the incremental effect of smoking on the levels of TNF-α in blood circulation (Bermudez *et al.,* 2002; Helmersson *et al.,* 2005). Confirming this argument, the literature indicates that smoking boosts production of inflammatory molecules in different cell types and leads to the incidence of systemic inflammation associated with increased inflammatory biomarkers such as TNF-α (Barbieri *et al.,* 2007; Frohlich *et al.,* 2003).

Scientific findings are inconsistent regarding the response of TNF-α to exercise in different healthy or sick populations. For example, in a recent study, 12 weeks of exercise significantly decreased serum CRP levels but caused no change in TNF-α in obese adult (Sharman *et al.,* 2004). Also some other studies failed to observe changes in the levels of TNF-α subsequent to training program compared with baseline levels (Miller *et al.,* 2008; Puglisi *et al.,* 2008). Yet another study found that levels of TNF-α would increase in skeletal muscle of lean elderly while exercise would decrease it (Greiwe *et al.,* 2001). In addition, a recent study showed that muscle contractions caused by aerobic - resistance exercise for 12 weeks would decrease the expression of cytokine inflammatory such as TNF-α at muscle levels, whereas no significant change in the levels of these cytokines has been observed in response to diet-induced weight loss (Charles *et al.,* 2008). Limited studies have so far been conducted on the response TNF-α to different types of exercise. This study aims to explore the effect of a three-month aerobic training program on serum levels of inflammatory cytokines in a group of middle-aged male smokers.

Materials and methods

Participants included 30 middle-aged smoker men aged 35 - 45 years and divided into exercise and control groups. Main objective of present study was to evaluate the effect of aerobic exercise program on serum TNF-α concentration in smoker men. All participants reported being weight stable for 6 months before study. The study was conducted with the approval of the Ethics Committee of Islamic Azad University. Inclusion criteria to study for smoker group were smoking history of At least 10 cigarettes a day for 3 years ago. Neither the control nor diabetic subjects had participated in regular exercise for the preceding 6 months. Potential participants were excluded from the study if they reported smoking or had a history of heart disease, stroke, or diabetes or were taking glucose-lowering medication. Those who that used lipid-lowering and hypertension medications were excluded.

After the nature of the study was explained in detail, all participants gave their informed written consent before participation. Body weight and height were measured with a standard physician's scale and a stadiometer, respectively when subjects were in a fasting state. Subjects were advised to be well hydrated and to limit their physical activity the day before the evaluation. Circumference measurements were taken with a Guilick Tape Measure to the closest 0.1 mm. Abdominal circumference was measured after a normal expiration using a non-elastic tape to

the nearest 0.1 cm. Body mass index was calculated as body mass (in kilograms) divided by height squared (in square meters). 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. Subjects were asked to attend Hematology Lab between the hours of 8 to 9 am. Blood samples were obtained in order to measuring serum TNF-α of each subject in two groups. Serum CRP was determined by ELISA method (Enzyme-linked Immunosorbent Assay for quantitative detection of human TNF-α). The Intra-assay coefficient of variation and sensitivity of the method were 6/0 % and 5/0 pg/mL respectively. All anthropometrical and biochemical measuring were repeated at 48 hours after lasted exercise session of aerobic exercise program.

Table 1. Mean and standard deviation of anthropometrical and serum TNF-a concentration before and after intervention in two studied groups.

Variables	Control Smokers		Exercise Smokers	
	Pretest	post-test	Pretest	post-test
Age (year)	41.3 ± 5.3	-------	41.8 ± 4.8	-------
Height (cm)	175.1 ± 6.7	-------	174.9 ± 6.5	-------
Weight (kg)	95.8 ± 5.4	94.5 ± 5.6	95.4 ± 4.5	91.2 ± 3.3
Abdominal circumference (cm)	108.5 ± 5.7	107.6 ± 6.4	108.7 ± 7.6	102.6 ± 5.6
Hip circumference (cm)	107.2 ± 6.3	106.6 ± 5.7	107.6 ± 7.8	101.3 ± 6.5
BMI (kg/m2)	31.24 ± 3.2	30.82 ± 3.4	31.51 ± 4.2	29.8 ± 2.3
Body fat (%)	31.5 ± 3.9	30.98 ± 3.5	31.33 ± 3.4	29.1 ± 3.4
Serum TNF-a (pg/ml)	37.21 ± 6.54	39.41 ± 4.21	38.33 ± 4.5	33.85 ± 5.33

Aerobic exercise program lasted 3 months (3 days/wk) 60 to 80 percent of maximum heart rate. Each session started by 15 min of flexibility exercises, 30-40 min of aerobic exercise and 5–10 min of cool down activity. Aerobic exercises in each session included walking on a treadmill and stationary cycling. The intensity of the activity of any person was controlled using the Polar heart rate tester (made in the US). In this 12-week period, participants in the control group were barred from participating in any exercise training.

Statistical analysis

Statistical analysis was performed with the SPSS software version 15.0. An Independent sample T-test was used to compare the serum levels of all anthropometrical and biochemical markers between smoker and non-smoker subjects. Student's t-tests for paired samples were performed to determine significance of changes in variables by intervention. All statistical tests were performed and considered significant at a $P \leq 0.05$.

Results

Table 1 presents fasting serum concentration of TNF-α and all anthropometrical markers in pre and post-training in exercise and control groups. There are no differences in body weight and body mass index between two groups at baseline. We also observed no significant differences in body fat percentage, abdominal and hip circumference between two groups. At baseline, serum TNF-α level were significantly higher in smokers in comparison to no-smoker subjects.

Determination of TNF-α response to aerobic exercise program was main objective of present study. In this regard, the data of Paired T test showed that aerobic exercise program was associated with improved serum TNF-a in exercise group but this inflammatory

cytokine did not change in control group. On the other hand, serum TNF-α was decreased significantly by exercise program in exercise group when compared to baseline ($p = 0.019$, Fig 1). Furthermore we observed a reduction in body weight in exercise subjects after treatment. Compared to pre-training, the other anthropometrical markers such as body mass index, body fat percentage and abdominal circumference were also significantly decreased ($p<0.05$) after exercise program in exercise group but not in control group.

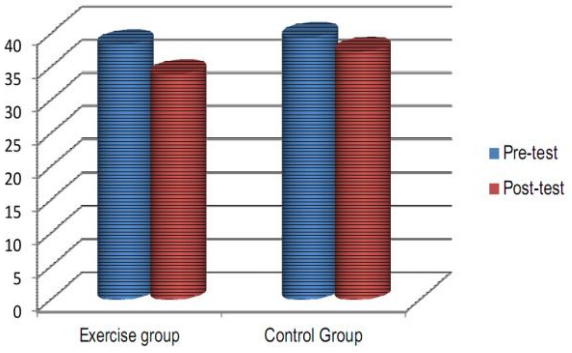

Fig. 1. The changes pattern of serum TNF-a concentration in control and exercise groups of studied subjects. This diagram shows that aerobic exercise program is associated with decreased serum TNF-a in exercise group, but this inflammatory cytokine did not change in control group

Discussion and conclusion

Most previous studies support higher levels TNF-α as an inflammatory cytokine in male smokers than non-smokers (Chung, 2001). In this study, a three-month aerobic training program significantly decreased TNF-α in experimental group. In other words, a three-month aerobic training with intensity of 60 to 80 percent of maximum heart rate three times per week significantly reduced serum levels TNF-α in adult male smoker. It is well known that smoking impairs the respiratory immunity function and increases the release of inflammatory mediators and ultimately leads to increased incidence of lung disease. Recent studies have shown that TNF-α plays an important role in smoking-related chronic obstructive pulmonary (COPD) disease and retains respiratory pathways inflammation (Chung, 2001). Moreover, experimental studies on mice have shown that tobacco use would increase levels of TNF-α in both serum and lung alveolar fluids (Pessina *et al.,* 1993; Pang *et al.,* 2000). These findings support inflammation of respiratory pathways in smokers and suggest increased risk of incidence of respiratory diseases such as COPD and asthma due to tobacco use (Merghani *et al.,* 2012). The findings of this study confirm that relatively moderate-intensity exercise for a long period of time is associated with reducing inflammation in healthy, obese or ill subjects. It has been established that the exercise affect differently levels of any of the inflammatory cytokines. Moreover, it should also be noted that methods of sampling and measurement also matter in inconsistency of the findings of the studies. It is known that smoking even a relatively low number of cigarettes significantly increases white blood cells count (Naoya *et al.,* 2011). The effect of smoking on the rise of WBC and TNF-α, however, is not yet fully determined because measuring TNF-α and its 1 and 2receptors is time consuming and costly. Nonetheless levels of body fat or body mass index can drastically affect WBC and TNF-α as it is difficult to measure the direct effect of smoking on WBC or such inflammatory cytokines as TNF-α. It seems that an all-encompassing conclusion can be reached as to the inflammatory cytokine response to aerobic exercise in male smokers only if the expression of these cytokines or tissue samples or pulmonary alveolar cells in are (Naoya *et al.,* 2011) measured in the respiratory tracts of the subjects.

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