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Anti-inflammatory effect of one moderate exercise in smokers

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Abstract

Tumour necrosis factor-alpha (TNF-α), a pro-inflammatory cytokines, is produced by human adipose tissue and it relation with cigarette smoking are not well understood. The aim of this study was to investigate immediate response of serum TNF-α to moderate exercise test fourteen non-athletes adult smoker men aged 40-48 years. For this purpose, venous blood sample was collected prior to exercise and immediately following of 35 min running at 70% of heart rate max. Pre- and post exercise serum TNF-α were compared using a paired-samples t-test. Significance was accepted at $P < 0.05$. Compared to pre-exercise, serum TNF-α decreased significantly ($p = 0.015$) after exercise test when compared to pretest. These data support anti-inflammatory effect of moderate exercise in smokers.

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Introduction

Smoking is undoubtedly one of the major public health problems. Tobacco use is well known to be important in increasing cardiovascular, respiratory and blood circulation diseases and its potential role in some cancers has frequently been reported. Meanwhile the effect of smoking on increased inflammatory cytokine levels or decreased anti-inflammatory cytokine levels that are particularly important in cardiovascular or respiratory disease has been observed in some recent studies (Yang *et al.*, 2006). In other words, active inflammatory cells produce and release a variety of inflammatory mediators in response to smoking, and the inflammatory cytokines are the most important of them. Importance of smoking in increasing inflammatory cytokines has been reported in some previous studies (Patrícia, 2011). Among pro-inflammatory cytokines, tumor necrosis factor (TNF) is the main chemical mediator of inflammatory responses in the face of negative bacterial infections and other infectious microbes and infections and is responsible for the symptoms of acute infections. Literature reports TNF-α as an inflammatory cytokine the plasma or serum levels of which are higher than normal in the presence of obesity or insulin resistance (Ye, 2008). The stimulatory role of TNF-α in increased production of vLDL has been observed in some studies which describes the relationship of this cytokine with TG Plasma (Qin *et al.*, 2008).

TNF-α is in fact, one of the most important cytokines secreted by adipose tissue and increase the presence of other inflammatory cytokine such as IL-6 and IL-1 in the blood (Weiss, 2005). Scientific findings about the response of TNF-α to exercise in healthy and ill populations are inconsistent. For example, in a recent study, 12 weeks of exercise led to a significant reduction in serum levels of CRP but to no change in TNF-α in obese adults (Sharman *et al.*, 2004). There are also other studies reporting no changes in the levels of TNF-α following a training program compare to baseline levels (Miller *et al.*, 2006; Puglisi *et al.*, 2008). But in another study it was found that levels of TNF-α would increase in skeletal muscles of lean elderly subjects, and exercise would decrease it (Greiwe *et al.*, 2001).

Despite the findings on the potential impact of inflammatory diseases on TNF-α some studies support the increasing effect of smoking on the circulatory levels of TNF-α (Bermudez *et al.*, 2002; Helmersson *et al.*, 2005). Confirming the above, the literature points out that smoking increases production of inflammatory molecules in different cell types and leads to the prevalence of systemic inflammation associated with an increase in inflammatory biomarkers, such as TNF-α (Frohlich *et al.*, 2003; Barbieri *et al.*, 2007). Given the limited studies on the response of TNF-α to exercise in smokers and also the contradictory findings in healthy or other ill populations in this area, this study aims to explain the response of this pro-inflammatory cytokine to a relatively long exercise session in male smokers.

Method and subjects

This study involved 14 non-athletes adult smoker men aged 40 - 48 years and height 172-180 cm. The objective of this investigation was estimate acute response of serum TNF-a to a long time exercise test with moderate intensity in mentioned subjects. This study was approved by the institutional ethical committee of Azad University, Iran. After the nature of the study was explained in detail, informed consent was obtained from all participants. An inclusion criterion was cigarette smoking for 5 years at least. Participants were included if they had not been involved in regular physical activity or diet in the previous 6 months. The exclusion criteria were infections, renal diseases, and hepatic disorders, use of alcohol and diabetes symptom.

Anthropometric measures

Body weight was measured in the morning following a 12-h fast. Height and body weight were measured twice, with subjects being barefoot and lightly dressed; the averages of these measurements were recorded. Body mass index (BMI) was calculated as

weight (kg)/ [height (m)]2.

Blood samples and exercise protocol

A venous blood sample was collected from all the subjects prior to exercise and immediately following exercise. Exercise protocol was same for all participants. Exercise test was 35 min running at 70% of heart rate max. Blood samples were analyzed for serum IL-6. Subjects were asked to avoid doing any heavy physical activity for 48 hours before blood sampling. After sampling in ETDA- or serum-tubes, blood was immediately chilled on ice, centrifuged and aliquots were frozen at – 80˚C until assayed. Plasma TNF-α concentration was determined using an Enzyme-linked Immunosorbent Assay for quantitative detection of human TNF-α. The intra-assay CV was 6.0%, and inter-assay variability ranged from 7.4%.

Statistical analyses

Statistical analysis was performed with the SPSS software version 15.0. All values are given as mean and standard deviation. For the descriptive statistics after having checked the normality of the variables using the Kolmogorov-Smirnov test. Pre- and post exercise serum IL-6 were compared using a paired-samples t-test. Significance was accepted at $P < 0.05$.

Results

Table 1 presents the anthropometric characteristics of the fourteen smoker subjects used.

Table 1. The physical characteristics of the subjects.

Variables	Age (years)	Weight (kg)	Height (cm)	BMI (kg/m²)	BF (%)
Mean and SD	44.7 ± 2.4	91.1 ± 7.94	177.5 ± 1.99	28.98 ± 2.56	28.47 ± 2.99

BMI, body mass index; BF, Body fat percentage.

Main objective of present study was to estimate serum TNF-a response to exercise test included 35 min running at 70% of heart rate max in smoker men. Data showed that exercise test led to significant decrease in this inflammatory cytokine in these subject (Pre to post: 56.57 ± 30.45 pg/ml, p = 0.015, Fig 1).

Discussion

Our study finding showed that exercise test was associated with significantly decrease in serum TNF-α in studied subjects. On the other hand, we observed that one session running test with relatively long-time at moderate intensity decreases serum TNF-α in adult smoker men. A growing body of evidence supports link between smoking and increased morbidity and mortality, and current trends indicate that of the one billion smokers worldwide, 500 million will die prematurely from smoking-related diseases (WHO, 2008).

Accumulating evidence indicates that cigarette smoking damages vascular endothelium which plays a fundamental role in the genesis of pathological cardiovascular events (Silvia *et al.*, 2011). Previous investigations have described an association Between smoking risks with changes in the expression of these cytokines (Lowe *et al.*, 2006; Vanderschueren *et al.*, 2006) emphasizing that short-term exposure to cigarette smoke in vivo is sufficient to increase IL-1b and/or TNF-a (Barbieri *et al.*, 2007; Castro *et al.*, 2004). It has recently been recognized that current smokers are characterized by increased white blood cell (WBC) counts and increased tumor necrosis factor-a (Taylor *et al.*, 1986; Petrescu *et al.*, 2010). On the other hand, It is known that cigarette smoking exposure to cigarette smoke increases levels of TNF-a in bronchial lavage fluid (Churg *et al.*, 2009) and in circulating mononuclear cells in smokers more than in non-smokers (Ryder *et al.*, 2002).

Although most recent studies support non-impact of short-term or single session of exercise on inflammatory cytokine levels and repeatedly emphasize the point that only those long-term exercises causing considerable body weight loss or

those single exercise sessions that lasts more than 60 minutes would lead to a significant reduction in serum levels of inflammatory cytokine (Hqjbjerre *et al.*, 2007; Kraemer *et al.*, 2000), but based on the findings of this study, perhaps it can be inferred that the response pattern of TNF-α in smokers is an exception to this rule, although the mere findings of this study cannot be considered definitive findings and to achieve more comprehensive results, further laboratory studies are needed.

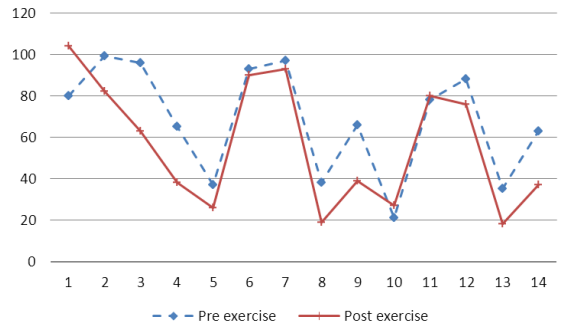

Fig. 1. This Fig indicate significant decrease of serum TNF-α by exercise test when compared with baseline in smokers subjects.

However, since the exercise test protocol in this study was of moderate rather than high intensity, it seems that implementing exercise protocols of relatively long duration with moderate intensity is associated with an anti-inflammatory effect in male smokers or other diseases. Also some studies somehow suggesting the anti-inflammatory effects of exercise argue that increased secretion and release of IL-6 by muscles active during exercise tests inhibit the secretion of TNF-α in the subjects, which supports the anti-inflammatory effects of exercise (Steensberg *et al.*, 2002; Steensberg *et al.*, 2000). In another study on non-smoker populations it was found that aerobic-resistance exercise-induced muscle contractions decrease the expression of inflammatory Cytokines such as TNF-α at muscle levels (Charles *et al.*, 2008). In a general summary, the findings of this study do not support the anti-inflammatory role of relatively moderate-intensity exercise of short duration on systemic inflammation profiles in male smokers although measuring TNF-α alone cannot represent all the effects of exercise on inflammation in smokers.

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