Antioxidative and Allergic Profile in Adult Men with Cigarette Smoking

Mozhgan Ahmadi*, Saeedeh Shadmehi and Mahdi Naji**

*Department of Physical Education and Sport Science, College of Management and Accounting, Yadegar-e-Imam Khomeini (RAH) Shahre-rey Branch, Islamic Azad University, Tehran, IRAN.

**Department of Physical Education and Sport Sciences, Islamshahr Branch, Islamic Azad University, Islamshahr, IRAN

(Corresponding author: Mozghan Ahmadi)
(Received 15 August, 2014, Accepted 14 September, 2014)

ABSTRACT: Accumulating experimental and epidemiologic data suggest that smoking is a central factor in many pathological conditions. The aim of this study was to determine if total antioxidant capacity (TAC) is related with serum Imonoglobin E (IgE) in cigarette smokers. For this purpose, serum IgE and TAC were measured after an overnight fast in adult males (n=30, age 41±4 yrs, body weight 89±4.9 Kg) with a history cigarette smoking at least for 3 years. Pearson’s correlation coefficients were used to evaluate the correlation between them. A p-value < 0.05 was considered to be statistically significant. Data showed a significant negative correlation serum IgE with TAC in studied subjects (P=0.011). These findings suggest that antioxidant system can be affect by IgE as an allergic agent in smokers.

Keywords: Ionomoglobin E, Total antioxidant capacity, Smoking

INTRODUCTION

According to the latest report published by the World Health Organization, one person per each 8 seconds dies of smoking in the world, 70% of whom are those who began smoking in their adolescence and continued this style of life for 20 years or more. If smoking continues in the same trend, it is expected that more than 9 million people, on average, annually will lose their lives directly because of smoking in 2030 [1]. Smoke, as one of the products of tobacco, affects various systemic organs through different diseases. Cardiovascular diseases and respiratory diseases such as chronic obstructive pulmonary and lung cancer are the major consequences of smoking [2, 3]. It has been known that smoking underlies the prevalence of many chronic and inflammatory diseases by suppressing the immune system and releasing inflammatory mediators [4, 5]. Furthermore, inhibition of anti-oxidative systems followed by smoking is also culpable for many pathological conditions mentioned above [6], because reduction and weakness in the performance of anti-oxidative system leads to the increased levels of free radicals and many oxidants [7]. According to available evidence, smoking increases free radicals and oxidants and decreases the total antioxidant capacity [8].

On the other hand, the literature suggests that smoking, through multiple mechanisms, increases the synthesis and release of IgE, paving the way for the emergence of atopic diseases and asthma [9]. Based on scientific studies, smokers have a higher level of IgE than non-smokers [10, 11].

It has been revealed that IgE (Ionomoglobin E) and mast cells play an important role in allergic inflammations. Scientific findings corroborate the fact that IgE is a key and effective factor in inflammatory reactions and plays an important role in pathogenesis of allergic diseases such as asthma [12]. The findings of previous studies indicate increased IgE levels and reduced total antioxidant capacity in response to smoking. However, the question is that whether these changes are the direct result of smoking or each of them also independently affects other levels. Hence, the present paper aims to answer the hypothesis that there is a significant relationship between total antioxidant capacity and serum IgE levels.

METHODS

A. Human Subjects

This study conducted on thirty adult men with cigarette smoking between 33 and 48 years old who voluntarily participated in this study. All study participants gave informed consent for the study by signing a form approved by Islamic Azad University, Iran. A detailed history and physical examination of each subject was carried out. Smoking 10 cigarettes a day for at least 5 years was main inclusion criteria. Participants were non-athletes and non-alcoholics. Participants were included if they had not been involved in regular physical activity/diet in the previous 6 months. Subjects with a history or clinical evidence of impaired fasting glucose or diabetes, recent myocardial infarction, active liver or kidney disease, the other chronic were excluded.
B. Anthropometric measures
Anthropometric measurements (body height and weight, waist and hip circumference) were performed with the subjects wearing light underwear and without shoes. Abdominal obesity was determined as waist circumference measured in a standing position. Weight was measured by an electronic balance and height by a stadiometer. Height was measured on standing while the shoulders were tangent with the wall. Body mass index (BMI) was calculated as weight (kg) divided by squared height (m).

C. Laboratory Analyses
To measure TAC and serum IgE, subjects asked to attend hematology lab after an overnight fast. Venous blood samples were obtained at rest between 8:00 and 9:00 am from the antecubital vein and Serum separated by centrifugation. The Intra-assay coefficient of variation and sensitivity of the method were 5.87% and 1.0 IU/mL, respectively for IgE (Monobind Inc, CA 92630, USA). TAC was determined by FRAP method (the sensitivity of method was 0.1 Units/ml).

D. Statistical analysis
Data were analyzed by computer using SPSS software version 15.0. Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. The association between serum IgE concentration and TAC were assessed using Pearson’s correlation coefficient. A P-value of < 0.05 was considered to be statistically significant.

RESULTS
Our objective of present study was to determine of relationship between serums IgE with total antioxidant capacity in adult smoker men. The physical and biochemical characteristics of the subjects are shown in Table 1.
Serum IgE was found to be negatively associated with total antioxidant capacity in studied subjects (p = 0.011, r = 0.46, Fig. 1). Between all anthropometrical markers, abdominal obesity was negatively correlated with total antioxidant capacity in subjects (p = 0.039, r = 0.38, Fig. 2).

| Table 1: Anthropometric and metabolic characteristics of the study participants |
|-------------------------------------------------|----------------|----------------|
| Variable                                      | Mean | Standard deviation |
| Age (years)                                   | 41   | 3.99             |
| Weight (kg)                                   | 86   | 4.9             |
| Height (cm)                                   | 173  | 2.8             |
| Abdominal circumference (cm)                  | 94   | 7.3             |
| Hip circumference (cm)                        | 96   | 5.8             |
| AHO ratio                                     | 1.00 | 0.05           |
| Body mass index (kg/m²)                       | 29.5 | 1.7             |
| Body Fat (%)                                  | 28.1 | 1.65           |
| Serum IgE (IU/mL)                             | 220  | 123             |
| Total antioxidant capacity (Units/ml)         | 0.31 | 0.19            |

Fig. 1: Relation of TAC with serum IgE in smoker. A negative correlation was observed between them.
DISCUSSION

A significant relationship was observed between serum IgE levels and total antioxidant capacity in the studied smokers. In other words, the findings of this study indicate that decrease in the total antioxidant capacity is followed by increased level of IgE, as an immunoglobulin stimulating allergy, in smokers. This supports the existence of a link between allergic stimuli and the antioxidant defense system in smokers.

Smokers are highly susceptible to cardiovascular diseases, chronic respiratory diseases, metabolic disorders, and diseases associated with metabolic syndrome such as hypertension, diabetes, and malignant diseases [13, 14, 15]. Studies have shown that long-term smoking leads to increase in inflammatory markers such as C-Reactive protein (CRP), Interleukin-6 (IL-6), Tumor necrosis factor alpha (TNF-α), Resistin, and Interleukin-1 beta (IL-1β), altogether cause and intensify chronic diseases including diabetes type 2, asthma, and cardiovascular diseases [16, 17, 18, and 19]. In addition, some previous studies indicate the direct relationship of smoking with levels of total cholesterol and low-density lipoprotein cholesterol and its inverse relationship with high-density lipoprotein cholesterol [20]. Although some changes caused by smoking are reversible after quitting it, some studies have reported that the levels of these inflammatory mediators stay high even after 10 to 20 years [17]. Some scientific references have stated that quitting smoking can delay the risk of cardiovascular disease by 5 to 10 years. However, the immediate effects of quitting smoking on inflammatory biomarkers associated with cardiovascular risk factors have not fully understood yet [21].

It has been found that level of lipid peroxidation is higher in smokers than non-smokers, which leads to atherosclerosis through the destruction of lipids [22]. It should be also noted that tobacco, for unknown reasons, reduces the antioxidant capacity of saliva [23]. Imbalance of the level of free radicals and reactive oxygen forms with antioxidants due to cigarette smoke could have a key role in the onset and development of inflammatory lesions in the mouth [24, 25, and 26]. Studies have shown that cigarette smoke is a major source of free radicals and tobacco smoke contains oxidation and peroxidation agents [27].

Extensive studies on the increased cardiovascular diseases in smokers mostly have emphasized on reduction in antioxidants levels and increase in oxidized lipid and lipoprotein levels followed by smoking [28, 29].

![Fig. 2](image)

**Fig. 2:** This Fig shows that TAC is negatively correlated with abdominal obesity in smoker.
On the other hand, smoking leads to a decline in buffering function of the pulmonary epithelium against allergens, as increased IgE resulting from smoking, as an allergic stimulus, leads to increased allergic inflammation or allergic diseases such as asthma [30]. Most studies in this regard have reported higher levels of IgE in smokers compared with non-smokers [10, 11]. The negative effects of smoking are such that even those who are exposed to cigarette smoke have higher levels of IgE than others [31].

Generally, the harmful effects of smoke lead to both decreased total antioxidant capacity and increased levels of IgE. These findings confirm the close relationship of allergic stimuli with oxidative stress and antioxidant defense system. It is also probable that the increase in free radicals and other oxidants, in response to a decrease in the total antioxidant capacity or reduced oxidative defense caused by smoking, leads to increased level of IgE that triggers allergic diseases like asthma. Some studies have supported the impact of both oxidative stress and mast cells in acute lung injuries [32]. It was concluded in a study that oxidative stress stimulates the expression of genes of some inflammatory cytokines in mast cells [33]. Since some studies have proved the direct relationship between IgE and inflammatory cytokines [34, 35], it seems that the increased expression of gene or increased levels of inflammatory cytokines in oxidative stress condition underlie an increase in secretion of IgE from these cells.

REFERENCES


