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Antioxidative and Allergic Profile in Adult Men with Cigarette Smoking

Mozhgan Ahmadi*, *Saeedeh Shadmehri* 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: Mozhgan Ahmadi)
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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: Imonoglobin 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 (Imonoglobin 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	Range
Age (years)	41	3.99	33 – 48
Weight (kg)	86	4.9	80 – 100
Height (cm)	173	2.8	167 – 179
Abdominal circumference (cm)	94	7.3	83 – 117
Hip circumference (cm)	96	5.8	86 – 108
AHO ratio	1.00	0.05	0.90 – 1.10
Body mass index (kg/m^2)	29.5	1.7	26.7 – 33.5
Body Fat (%)	28.1	1.65	25.3 – 32.3
Serum IgE (IU/mL)	220	123	69 – 473
Total antioxidant capacity (Units/ml**)**	0.31	0.19	0.12 – 0.93

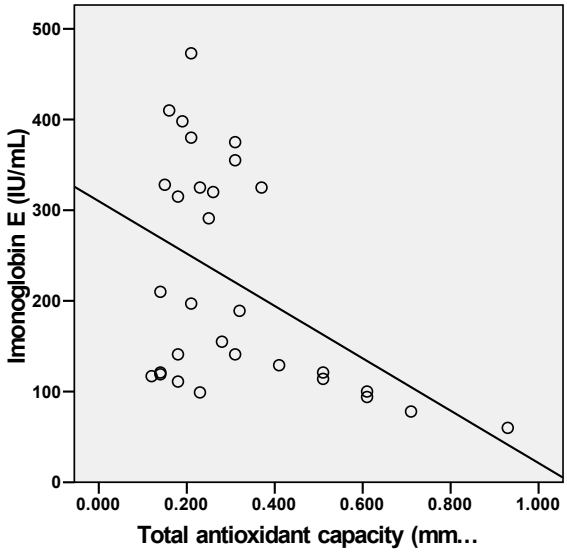

Fig. 1: Relation of TAC with serum IgE in smoker. A negative correlation was observed between them.

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-1ß), 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 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].

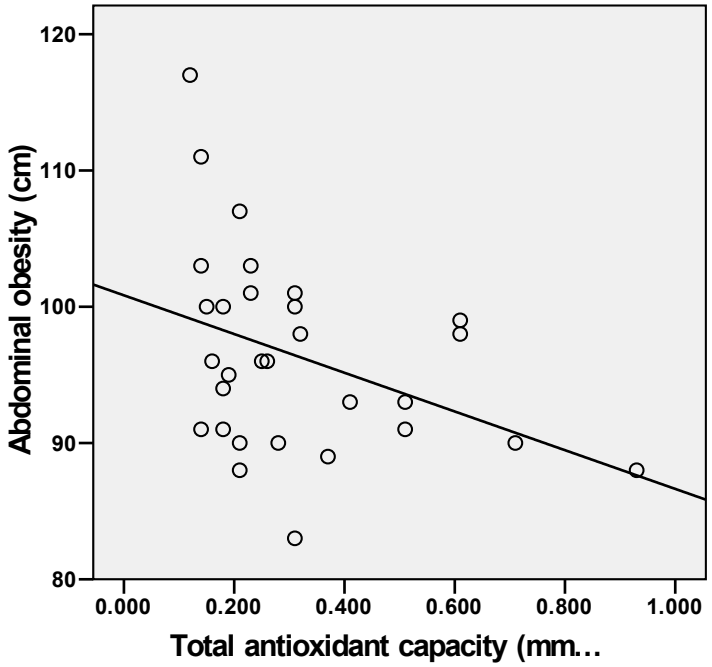

Fig. 2: This Fig shows that TAC is negatively correlated with abdominal obesity in smoker.

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

World health report (2002): Reducing risks, promoting healthy life. Geneva, Switzerland: World Health Organization.

Crofton J., Bjartveit K. (1989). Smoking as a risk factor for chronic airways disease. *Chest* **96**(3 suppl): 307–312.

Boyle P. (1997). Cancer, cigarette smoking and premature death in Europe: a review including the Recommendations of European Cancer Experts Consensus *Meeting, Helsinki, October 1996. Lung Cancer* **17**: 1–60.

Walters M.J., Paul-Clark M.J., McMaster S.K. (2005). Cigarette smoke activates human monocytes by an oxidant-AP-1 signaling pathway: implications for steroid resistance. *Mol Pharmacol* **68**: 1343–1353.

Barbieri S.S., Weksler B.B. (2007). Tobacco smoke cooperates with interleukin-1ß to alter ß -catenin trafficking in vascular endothelium resulting in increased permeability and induction of cyclooxygenase-2 expression in vitro and in vivo. *FASEB J* **21**:1831–1843.

Rao G.M., Sumita P., Roshni M., Ashtagimatt M.N. (2005). Plasma antioxidant vitamins and lipid peroxidation products in pregnancy induced hypertension. *Indian Journal of Clinical Biochemistry.* **20**(1): 198-200.

Rouzbahani R, Asgary S, Naderi GA, Dehghan Nejad M, Rezaei F. (2009). Comparision of plasma peroxidants, glycosilated hemoglobin, conjugated dienes and CRP level in smokers and non-smokers men. *Journal of Isfahan Medical School.* **27**(93): 115-121.

Polidori MC, Mecocci P, Stahl W, Sies H. (2003). Cigarette smoking cessation increases plasma levels of several antioxidant micronutrients and improves resistance towards oxidative challenge. *Br J Nutr* **90**: 147-150.

Arnson Y, Shoenfeld Y, Amital H. (2010). Effects of tobacco smoke on immunity, inflammation and autoimmunity. *J Autoimmun.* **34**(3): 258-65.

Lim HB, Kim SH. (2014). Inhallation of e-Cigarette Cartridge Solution Aggravates Allergen-induced Airway Inflammation and Hyper-responsiveness in Mice. *Toxicol Res*. **30**(1): 13-8.

Hizume DC, Toledo AC, Moriya HT, Saraiva-Romanholo BM, Almeida FM, Arantes-Costa FM, Vieira RP, Dolhnikoff M, Kasahara DI, Martins MA. (2012). Cigarette smoke dissociates inflammation and lung remodeling in OVA-sensitized and challenged mice. *Respir Physiol Neurobiol.* **181**(2): 167-76.

Nowak D. (2006). Management of asthma with anti-immunoglobulin E: A review of clinical trials of omalizumab. *Respiratory Medicine*. **100**: 1907–1917.

Axelsen M, Eliasson B, Joheim E, Lenner RA, Taskinen MR, Smith U. (1995). Lipid intolerance in smokers. *J Intern Med.* **237**(5): 449-455.

Bruckert E, Jacob N, Lamaire L, Truffert J, Percheron F, de Gennes JL. (1992). Relationship between smoking status and serum lipids in a hyperlipidemic population and analysis of possible confounding factors. *Clin Chem.* **38**(9): 1698-1705.

Eliasson B, Mero N, Taskinen MR, Smith U. (1997). The insulin resistance syndrome and postprandial lipid intolerance in smokers. *Atherosclerosis.* **129**(1): 79-88.

Arnson Y, Shoenfeld Y, Amital H. (2010). Effects of tobacco smoke on immunity, inflammation and autoimmunity. *J Autoimmun.* **34**(3): 258-65.

Dilyara G. Yanbaeva, Mieke A. Dentener, Eva C. Creutzberg, Geertjan Wesseling and Emiel FM. Wouters. (2007). Systemic Effects of Smoking. CcChest. 1557-1566.

Orosz Z., Csiszar A., Labinskyy N., Smith K., Kaminski P.M., Ferdinandy P., Wolin M.S., Rivera A., Ungvari Z. (2007). Cigarette smoke-induced proinflammatory alterations in the endothelial phenotype: role of NAD (P) H oxidase activation. *Am J Physiol Heart Circ Physiol.* **292**(1): 130-9.

Feskens E.J., Kromhout D. (1989). Cardiovascular risk factors and the 25-year incidence of diabetes mellitus in middle-aged men. The Zutphen Study. *Am J Epidemiol* **130**: 1101–1108.

Tsujii S., Kuzuya H. (2004). The significance of lifestyle as a factor for the metabolic syndrome. *Nippon Rinsho.* **62**(6): 1047-1052.

Virginia R., Xiangying X., Diane B., Daniel J., Christine F., Patricia F. (2011). A Pilot Study to Examine the Effects of Smoking Cessation on Serum Markers of Inflammation in Women at Risk for Cardiovascular Disease. *Chest.* 212-219.

Henriksen T., Mahoney E.M., Steinberg D. (1983). Enhanced macrophage degradation of biologically modified low density lipoprotein. *Arteriosclerosis* **3**(2): 149-59.

Battino M., Ferreiro M.S., Gallardo I., Newman H.N., Bullon P. (2002). The antioxidant capacity of saliva. *J Clin Periodontol* **29**(3): 189-94.

Pasupathi P., Rao Y.Y., Farook J., Saravanan G., Bakthavathsalam G. (2009). Effect of cigarette smoking on lipids and oxidative stress biomarkers in patients with acute myocardial infarction. *Res J Med Sci* **4**(2): 151-9.

Preston A.M. (1991). Cigarette smoking-nutritional implications. Prog Food Nutr Sci **15**(4): 183-217.

Kosecika M., Erelb O., Sevincc E., Selekb S. (2005). Increased oxidative stress in children exposed to passive smoking. *Int J Cardiol* **100**(1): 61-4.

Yildiz L., Kayaoˇglu N., Aksoy H. (2002). The changes of superoxide dismutase, catalase and glutathione peroxidase activities in erythrocytes of active and passive smokers. *Clin Chem Lab Med* **40**(6): 612-5.

Miller E.R., III, Appel L.J., Jiang L., Risby T.H. (1997). Association between cigarette smoking and lipid peroxidation in a controlled feeding study. *Circulation* **96**(4): 1097-101.

Schooler C., Feighery E., Flora J.A. (1996). Seventh graders' self-reported exposure to cigarette marketing and its relationship to their smoking behavior. *Am J Public Health* **86**(9): 1216-21.

Gangl K., Reininger R., Bernhard D., Campana R., Pree I., Reisinger J., Kneidinger M, Kundi M, Dolznig H., Thurnher D., Valent P., Chen K.W., Vrtala S., Spitzauer S., Valenta R., Niederberger V. (2009). Cigarette smoke facilitates allergen penetration across respiratory epithelium. *Allergy*. **64**(3): 398-405.

Bahna S.L., Heiner D.C., Myhre B.A. (1983). Immunoglobulin E pattern in cigarette smokers. *Allergy*. **38(1):**57-64.

Zhao W., Gan X., Su G, Wanling G, Li S, Hei Z, Yang C, Wang H. (2014). The interaction between oxidative stress and mast cell activation plays a role in acute lung injuries induced by intestinal ischemia-reperfusion. *J Surg Res.* **187**(2): 542-52.

Frossi B., De Carli M., Daniel K.C., Rivera J., Pucillo C. (2003). Oxidative stress stimulates IL-4 and IL-6 production in mast cells by an APE/Ref-1-dependent pathway. *Eur J Immunol.* **33**(8): 2168-77.

Eizadi M., Dooaly H., Kiani F. (2014). The association between serum IL-1ß and IgE in healthy obese men. *Advances in Bioresearch***. 5**(2): 1-4.

Thomas S.S., Chhabra S.K. (2003). A study on the serum levels of interleukin-1beta in bronchial asthma. *J Indian Med Assoc*. **101**(5): 282-286.