Biological Forum – An International Journal

6(2): 504-508(2014)

ISSN No. (Print): 0975-1130 ISSN No. (Online): 2249-3239

Serum Imonoglobin E and aerobic training in cigarette smoke

Sokhanguei Yahya, Masoomeh Edrisi and Mohammadi Rasoul

Department of Physical Education and Sport Sciences, Central Tehran Branch, Islamic Azad University, Tehran, IRAN

(Corresponding author: Sokhanguei Yahya) (Received 27 September, 2014, Accepted 30 October, 2014)

ABSTRACT: Serum IgE is known to be influence in allergic or asthma prevalence. The objective of this study was to evaluate the effect of aerobic training program on serum IgE in cigarette smokers. A totally 24 sedentary adult smoker males aged 36 ± 12 year and height 174 ± 4 cm were participated in this study by accessible samples and selected into exercise and control groups by randomly. Inclusion criteria for participate in study was smoking 10 cigarettes a day for at least 5 years. Pre and post training (3 months aerobic training, 3 times / weekly) blood samples were collected of all participants in two groups on order to calculation serum IgE. Student's t-tests for paired samples were performed to determine significance of change in serum IgE by aerobic program. P value of <0.05 was accepted as significant. These were no significant difference in serum IgE between pre and post training in exercise group. Aerobic training resulted in significant decrease in all anthropometrical markers. All variables remained without change in control subjects. Based on these data, it concludes that long term aerobic training can not affect serum IgE in cigarette smokers.

Keywords: Allergy, Aerobic Training, Smoking, Inflammation

INTRODUCTION

Not only for years or consecutive decades, but it is for centuries that smoking has become prevalent, such that it is more than 4 centuries that consuming tobacco, as a daily habit, has rooted in everyday human life. Perpetual consumption of tobacco has been known as the second leading cause of death in today's world. Secreting inflammatory mediators into the bloodstream by smoking has been introduced as the cause of many chronic and inflammatory diseases [1, 2].

The inflammatory effects of smoking are reported so high that even after 10 to 20 years after quitting smoking, some inflammatory mediators, like C-Reactive protean, still remain in high levels in the body. Although some changes due to smoking are reversible after quitting [3]. New statistics and numbers indicate that more than 700 million children suffer direct inhaling of cigarette smoke [4, 5].

It has been clear that smoking increases the activity of many cells and stimulates inflammatory mediators and markers indicating the decreases performance of the immune system. For instance, in a relatively recent study, it was pointed out that smoking can mitigate the buffering performance of pulmonary epithelium against some allergens and can increase allergic inflammatory and allergic diseases by increasing the synthesis and secretion of immunoglobulin E (IgE) into the bloodstream [6]. Similar to other immunoglobulins, IgE is produced by plasma cells and B cells. Due to the high absorption power of mast cells on IgE, IgE levels of blood circulation is relatively lower in comparison to other immunoglobulins. IgE and mast cells play an important role in the immune system. The role of IgE and the activities of mast cells in asthma are clear through the close connection of increased serum levels of IgE and the intensity of asthma [7].

Scientific references indicate that tobacco and smoking lead to increased density of IgE through multiple mechanisms which provide the condition for the spread of atopic diseases and asthma [8]. Therefore, it seems that reducing or quitting smoking or performing interventions or external stimuli, for instance, participating in exercise programs, particularly long term sport activities, lead to mitigating the side-effects of smoking. Based on this assumption, this study aims to determine the effect of a three month aerobic exercise on serum levels of IgE in a group of male smokers.

METHOD AND SUBJECTS

This study aimed to assess the effect of aerobic training on serum IgE in smokers. Subjects were twenty four non-trained males matched for age $(36 \pm 12 \text{ years})$ and height $(174 \pm 4 \text{ cm})$ with a history of cigarette smoking at least three years that participate in study through an advertisement in a local newspaper. Subjects were selected into exercise (n=12) or control (n=12) randomly. The exercise subjects were completed a three months aerobic training and control subjects were inactive in this period. Written consent was obtained from each subject after the experimental procedures and possible risks and benefits were clearly explained.

Inclusion criteria: Participants were non trained and non-alcoholic. All participants reported being weight stable $(\pm 1 \text{kg})$ for 6 months prior to the study and engaged in physical activity less than once per month. Having history of at least 10 cigarettes a day for 5 years was the main criterion for inclusion. Those with known history of respiratory infections, neuromuscular disease, cardiopulmonary disease and type II diabetes or other chronic diseases were excluded.

Anthropometry: Before and after the aerobic training, anthropometrical markers were measured in the morning following a 12-h fast. Height (Ht) and weight (Wt) were measured twice to ± 0.2 cm and to ± 0.2 kg, respectively, with subjects being barefoot and lightly dressed; the averages of these measurements were recorded. BMI was calculated by dividing body mass (kg) by height in meters squared (m2). Waist to hip ratio (WHR) was calculated as waist circumference divided by hip circumference. Waist circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter.

Biochemistry and exercise protocol: Pre and post training blood samples were collected of all participants in two groups on order to calculation serum IgE.

Venous blood samples were obtained at rest between 8:00 and 9:00 am from the antecubital vein and Serum separated by centrifugation. Blood was drawn after 12 h of fasting and 1 day of minimal physical activity. The Intra- assay coefficient of variation and sensitivity of the method were 5.8% and 1.0 IU/ml respectively. Aerobic training program lasted three months for three sessions per week. Each exercise session involved 5-10 warm up, 30-45 main exercise and 5-10 cool up. Main exercise was walking or running on treadmill or smooth surface with no slope in a range of intensity between 60–80 (%) of maximal heart rate. Control subjects were instructed to maintain their habitual activities.

Participants were instructed to maintain their usual diet throughout the duration of the study.

Statistical analysis: The data were reported as mean and standard deviation, and analyzed using the SPSSW statistical package, version 16.0)SPSS Inc., Chicago, IL, USA) for Windows W. Normality of distribution was assessed by Kolmogorov–Smirnov test. Independent student t test was used for between groups comparison. Pre- and post exercise serum IL-6 and anthropometrical markers were compared between conditions using a paired-samples t-test. Significance was accepted at P < 0.05.

RESULTS AND DISCUSSION

Table 1 show the descriptive anthropometric and biochemical features of the exercise subjects. Anthropometric and biochemical characteristics of the control subjects are also described in Table 2. Compared to pre-training, serum IgE concentration did not change by exercise program when compared with baseline (p = 0.839, Table 3, 4). This variable was also remained without change in control groups. Aerobic exercise program results in significant decrease in body weight (p = 0.018), BMI (p = 0.017), Abdominal circumference (p = 0.000), hip circumference (p =(0.000) and body fat percentage (p = (0.000)) (Table 3 and 4). The research findings regarding the response of IgE to different short and long term exercise programs are contradictory. This study was also unsuccessful to find a significant change in IgE by doing exercises. In other words, the three month aerobic exercise, three sessions per week, made no effect on the IgE levels of male smokers which lived an inactive life before.

The smokers are facing high risks of developing cardiovascular diseases, chronic respiratory diseases, metabolic diseases and diseases related to the metabolic syndrome, e.g. blood pressure, diabetes, and malignant diseases [9, 10, 11]. Scientific references have introduces factors like oxidative stress injury due to smoking as one of the most important factor of developing such anomalies in smokers [12, 13]. On the other hand, the degree of its synthesis by its constructive cells is relatively lower. It has been shown that the IgE levels are mostly increased under allergic conditions and its increase consequently intensifies allergic, inflammatory and infectious diseases. Particularly in industrialized countries, allergy is the most common reason for its increase. On the other hand, parasite infections have been introduced as the most common of its spread in developing countries [14].

	Minimum	Maximum	Mean	Std. Deviation
Age (year)	12	50	36.18	12.197
Height (cm)	168	180	174.36	3.613
Weight (kg)	83	99	92.00	5.495
Abdominal (cm)	96	115	102.36	5.353
Hip (cm)	95	106	100.27	3.823
BMI (kg/m2)	27.41	35.08	30.2855	2.05217
Body fat (%)	26.3	32.4	29.218	2.1558
Immonoglobin E (IU/mI)	60	473	218.36	122.401
WBC	5200	9700	7342.86	1897.241
RBC	4.7	5.3	5.117	.2049
НВ	14.4	16.7	15.514	.8611
Hct	42.6	50.4	45.429	3.0680
MCV	81.1	94.4	88.771	4.8784
MCH	27.4	32.1	30.329	1.6879
MCHC	33.1	35.1	34.171	.7610
PLT	204000	350000	269285.7	56227.853
LYMPH%	27.1	38.2	33.857	3.4674

Table 1: Descriptive Statistics of anthropometrical and clinical markers of exercise subjects at baseline

le 2: Descriptive Statistics of anthropometrical and clinical markers of control subjects at baseline

	Minimum	Maximum	Mean	Std. Deviation
Age (year)	38	48	42.18	3.516
Height (cm)	170	178	174.09	2.300
Weight (kg)	85	99	92.55	5.520
Abdominal (cm)	94	116	102.82	7.040
Hip (cm)	94	109	102.64	4.965
BMI (kg/m2)	28.08	33.91	30.5364	1.74340
Body fat (%)	26.7	33.1	29.382	1.8925
Immonoglobin E (IU/mI)	125	411	212.82	86.702
WBC	5300	9700	7414.29	1694.529
RBC	5.0	5.5	5.291	.1655
НВ	14.1	16.1	15.271	.7088
Hct	40.5	47.6	44.457	2.6248
MCV	76.6	88.4	84.086	4.4510
MCH	26.7	30.0	28.886	1.1510
MCHC	33.4	35.5	34.371	.8220
PLT	219000	336000	294142.9	39813.853
LYMPH%	26.2	45.9	35.943	6.6898

Variables	Exercise subjects			
	Pretraining	post-training		
Weight (kg)	92 ± 5.49	88.9 ± 5.61		
Waist circumference (cm)	102 ± 5.4	98 ± 4.8		
Hip circumference (cm)	100 ± 3.8	95.6 ± 4.1		
BMI (kg/m2)	30.28 ± 2.05	29.26 ± 1.96		
Body fat (%)	29.2 ± 2.15	27.7 ± 1.87		
Serum IgE (IU/ml)	218 ± 122	210 ± 133		

Table 3: Mean and standard deviation of anthropometrical and serum IgE in exercise groups.

Active smokers and people exposes to cigarettes have a higher IgE level than non-smokers [15]. It has been shown that smoking increases the production and secretion of inflammatory cytokines including TNF-, IL-6, IL-8, and IL-1, and reduces IgE levels which indicate its inflammatory effects [8]. Moreover, increased secretion of IgE from mast cells is another harmful agents of smoking, since it provides the conditions for developing or intensifying asthma and atopic diseases [8, 16]. It has been shown that even in non-smokers, who are exposed to cigarette smoke, have increased IgE levels [17]. IgE and mast cells play an important role in the immune system. The role of IgE and activates of mast cells in asthma is clear through the close connection of increased serum levels of IgE and the intensity of asthma [7].

Regarding the findings of this study and previous research, it seems that the type and protocol of the exercise program has a determinant role in IgE responses. In line with our findings, despite losing weight in response to a three month sport exercise, another study has found no significant change in IgE levels of obese men [18]. This research indicates that IgE levels of obesity, which is an effective factor in developing allergy and asthma, is considerably higher than people with normal weights [20]. The fat mass is a predictor of IgE levels in obese people [21]. The study of Thomas (2003) discusses the important role of IL-1ß and IgE in obesity related asthma diseases [22]. Researchers have supported the direct and significant connection of IL-1ß and IgE in the obese population [23]. Furthermore, the type of the studies population and the initial levels of IgE is very important in its response to exercise. Another study shows that one biking session leaded to a significant reduction of IgE in asthma patients [24].

In summation, it generally seems that several factors are effective in IgE levels of smokers or other healthy or patient populations. According to previous observations, it can be concluded that the response of IgE to exercise is related to its type, intensity, and duration, as well as the studies population. On the other hand, we must point out that the initial IgE levels, i.e. its resting values, before the exercise also play an important role in its later response to external interventions.

REFERENCES

- Walters MJ, Paul-Clark MJ, McMaster SK. (2005). Cigarette smoke activates human monocytes by an oxidant-AP-1 signaling pathway: implications for steroid resistance. *Mol Pharmacol.* 68:1343–1353.
- Barbieri SS, Weksler BB. (2007). Tobacco smoke cooperates with interleukin-1β to alter β 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.
- Dilyara G. Yanbaeva, Mieke A. Dentener, Eva C (2007). Systemic Effects of Smoking. *CcChest.* 14: 1557-1566.
- Ekerbicer HC, Celik M, Guler E, Davutoglu M, Kilinc M. (2007). Evaluating environmental tobacco smoke exposure in a group of Turkish primary school students and developing intervention methods for prevention. *BMC Public Health.* 7: 202.
- Hwang SH, Hwang JH, Moon JS, Lee DH. (2012). Environmental tobacco smoke and children's health. *Korean J Pediatr.* **55**: 35-41.
- Gangl K, Reininger R, Bernhard D, Campana R, Pree I, Reisinger J, Kneidinger M. (2009).Cigarette smoke facilitates allergen penetration across respiratory epithelium. *Allergy.* 64(3):398-405.
- Burrows B, Martinez FD, Halonen M, Barbee RA & Cline MG. (1989). Association of asthma with serum IgE levels and skin-test reactivity to allergens. *New England Journal of Medicine*. **320**: 271-277.

- Arnson Y, Shoenfeld Y, Amital H.(2010). Effects of tobacco smoke on immunity, inflammation and autoimmunity. J Autoimmun. 34(3): J258-65.
- 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.
- Lodovici M, Casalini C, Cariaggi R, Michelucci L, Dolara P. (2000). Levels of 8hydroxydeoxyguanosine as a marker of DNA damage in human leukocytes. *Free Radic Biol Med.* **28**(1): 13-17.
- Yamaguchi Y, Haginaka J, Morimoto S, Fujioka Y, Kunitomo M. (2005). Facilitated nitration and oxidation of LDL in cigarette smokers. Eur J Clin Invest. **35**(3): 186-193.
- Winter WE, Hardt NS, Fuhrman S. (2000). Immunoglobulin E, importance in Parasitic Infections and Hypersensitivity Responses. *Arch Pathol Lab Med.* **124**(9): 1382-5.
- Bahna SL, Heiner DC, Myhre BA. (1983). Immunoglobulin E pattern in cigarette smokers. *Allergy*. **38**(1):57-64.
- Lim HB, Kim SH. (2014). Inhallation of e-Cigarette Cartridge Solution Aggravates Allergeninduced Airway Inflammation and Hyperresponsiveness in Mice. *Toxicol Res.* **30**(1): 13-8.

- Hizume DC, Toledo AC, Moriya HT, Saraiva-Romanholo BM, Almeida FM, Arantes-Costa FM. (2012). Cigarette smoke dissociates inflammation and lung remodeling in **OVA-sensitized** and challenged mice. Respir Physiol Neurobiol. **181**(2): 167-76.
- Eizadi M, Kiani F, Dooaly H. (2014). Aerobic training program for long term does not anti-allergic property in obese subjects. *Indian Journal of Fundamental and Applied Life Sciences*. **4**(1): 390-394.
- IgE and IgG4 Immune Reactions to Food. www.metametrix.com/.../IgE-and-IgG4-Immune-Reactions-to-Food-0509.pdf.
- Visness CM, London SJ, Daniels JL, Kaufman JS, Yeatts KB, Siega-Riz AM. (2009). Association of obesity with IgE levels and allergy symptoms in children and adolescents: results from the National Health and Nutrition Examination Survey 2005-2006. J Allergy Clin Immunol. **123**(5): 1163-9.
- Vieira VJ, Ronan AM, Windt MR, Tagliaferro AR. (2005). Elevated atopy in healthy obese women. *Am J Clin Nutr.* **82**(3): 504-9.
- Thomas SS, Chhabra SK. (2003). A study on the serum levels of interleukin-1beta in bronchial asthma. J Indian Med Assoc. **101**(5):282-286.
- Eizadi M, Dooaly H, Kiani F. (2014). Single bout exercise is associated with allergic property in healthy obese men. *Indian Journal of Fundamental and Applied Life Sciences*. **4**(1): 226-229.
- Eizadi M, Shafiei M, Rohani AA, Jenabi A. (2011). Exercise as a none-pharmologicel intervention in maintains IgE in asthma patients. *Indian Journal of Fundamental and Applied Life Sciences*. 2011; **1**(4): 371-375.