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Hs.C-reactive protein is affected by long term aerobic exercise in asthma subjects

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Abstract

The aim of this study was to evaluate the effects of exercise training on serum CRP levels in adult asthma patients. For this purpose, pre and post aerobic training (3 times-weekly/ 3 months) blood samples were taken after overnight fast in 32 adult men with asthma patients that divided to exercise and control groups by randomly. Anthropometrical indexes were also monitored in two occasions. All data changes were compared by T tests. At baseline, there were no differences in the serum CRP, body weight and other anthropometrical indexes between the two groups. Compared to pre-training, the CRP levels decreased significantly after aerobic training in the exercise group but not in the control subjects. We observed a significant decrease in al anthropometrical indexes in exercise group. All paper remained without change in control group. Based on these data, we can say that exercise training for long time improves systemic inflammation in asthma patients.

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Introduction

To date, the current management of asthma focuses on the optimal control of airway inflammation as a central component of asthma control. Asthma is a disorder characterized by inflammation of the airways an inflammation cytokines play an important role in this disease (Heidenfelder *et al.,* 2010). Accumulating evidence indicates that asthma is a complex syndrome with many clinical phenotypes. Its major characteristics include a variable degree of airflow obstruction, bronchial hyper-responsiveness and chronic airway inflammation. There is considerable evidence that circulating CRP has emerged as one of the most powerful independent predictors of cardiovascular disease risk and cardiovascular death (Tice, 2003).

It has been long known that C-reactive protein (CRP) is a major inflammation sensitive plasma protein in humans and its synthesis by the liver is regulated to a large extent by the pro-inflammatory cytokine interleukin (IL)-6. It was reported that after adjustment for age, smoking status, diabetes, blood pressure and the use of hormone replacement therapy, the relative risk in top CRP is more than anthers cardiovascular risk factors such as LDL (Ridker, 2000). A large body of evidence suggests that Systemic inflammation is a possible element in the link between respiratory impairment and cardiovascular events. It was also reported that reduced lung function has been associated with various inflammation sensitive plasma proteins (Kony *et al.,* 2004; Mendall *et al.,* 2000).

In this area, some researchers suggests that systemic inflammation, as measured by using C-reactive protein (CRP) levels, might be important in the relationship between obesity and asthma (Butland *et al.,* 2008). Several studies have found a high inverse relation between CRP and function such as forced expiratory volume in one second (FEV1) (O'Connor *et al.,* 1995; Rijcken *et al.,* 1995). Some studies have indicated a positive correlation between asthma and increased CRP levels (Ebrahim *et al.,* 2011; Ford, 2003; Jousilahti *et al.,* 2002; Olafsdottir *et al.,* 2005). There are few longitudinal studies that physical activity or aerobic training decreases systemic inflammation in obesity or chronic diseases (de Salles *et al.,* 2010; Sheu *et al.,* 2008). But, there is limited literature on CRP responses to exercise training in asthma patients. Therefore, the present study aims to evaluate the significance of a long term aerobic exercise on serum CRP in adult men with asthma.

Material and methods

The study was approved by the Ethics Committees of Islamic Azad University, Parand branch. The objective of this semi-experimental study was to evaluate serum CRP response to aerobic exercise program in group asthma patients. For this purpose, thirty two adult men with asthma (39±5 years, body mass index 29.06±3 Kg) participated in this study by accessible sampling and divided to exercise (3 days/week for 12 weeks) and control (no exercise) groups by randomly. Spirometry tests (Minispire model, Made in Italy) was performed in order to asthma diagnosis as well as to determine the asthma severity. FEV1 and forced expiratory volume in 1 s / forced vital capacity (FEV1/FVC) were measured. All patients were asked to avoid having tea or coffee as well as other airways dilator food for at least 4 hours prior to spirometry test. Each participant received written and verbal explanations about the nature of the study before signing an informed consent form. All subjects were inactive, and none reported engaging in systemic (more than one time per week) sport activities before the study. Inclusion criteria for study group were determined as existing asthma for at least 2 years. Exclusion criteria included medications that alter carbohydrate metabolism, diabetes, inability to exercise, and history of hypertension or heart disease.

Pre and post training anthropometrical measurements and blood samples were completed in all patients of 2 groups. Measurements of height (m) and weight (kg)

were performed with the barefoot and dressed in shorts and shirt. With these measures, the body mass index (BMI = weight/height²) was calculated. Visceral fat and body fat percentage was determined using body composition monitor (OMRON, Finland). The subjects were advised to avoid any physical activity or exercise 48 hours before the blood sampling. Blood samples were taken between 8:00 and 9:00 a.m. after 10 to 12 hours overnight fast to measure serum CRP. A 5 ml fasting blood samples were collected from brachial vein in sitting position. Serums were immediately separated and stored at -80° until the assays were performed. In fact, fasting blood samples were taken pre-training (pre-test) and 48 h after aerobic exercise training program(post-test). Serum CRP was determined by ELISA method (Diagnostics Biochem Canada Inc. High sensitivity C - reactive protein (Hs-CRP)). Exercise training program was performed 60 to 80 percent of maximum heart rate. Aerobic exercises included walking or running and stationary cycling. The intensity of the activity of any person was controlled using the Polar heart rate tester. All participants in the control group were barred from participating in any exercise training. Finally, all measurements consist of fasting blood sampling; anthropometric measurements repeated 48 h after last exercise session.

Statistical analysis

Statistical analysis was performed with the SPSS software version 16.0. The Kolmogorov-Smirnov test was applied to determine the variables with normal distribution. Independent sample t test used to observe group mean difference at baseline. Pre- to post training changes were determined by two-tailed t tests. A p-value < 0.05 was considered to be statistically significant.

Results

Baseline and post training CRP levels and anthropometrical indexes of two groups are shown in Table 1. the spirometric test showed that all patients in mild to moderate asthma severity (FEV1 = 74 ± 6.5 (%), FEV1 / FVC = 67.3 ± 4.2). The finding by independent T test showed no differences in the age, spirometry markers, body weight and other anthropometrical indexes and serum CRP between the two groups ($p \leq 0.05$). Compared to pre-training, serum CRP decreased significantly ($p = 0.011$) after exercise program but not in the control groups (Fig 1). This finding indicates positive role of aerobic exercise training of CRP an inflammatory cytokine. Anthropometrics variables improved significantly after the therapy in exercise group. Body mass index levels were significantly decreased in response to exercise program when compared with baseline levels ($P = 0.021$). In addition, we found that aerobic exercise program reduced body fat percentage, body weight and visceral fat in exercise group ($p > 0.05$).

Discussion

Our study showed that aerobic exercise training for three months decreases serum CRP in adult men with asthma patients. In present study, we also observed that a reduction in anthropometric variables after aerobic training in exercise group. Adipose tissue secretes a variety of bioactive mediators including adipocytokines such as adiponectin, leptin, resistin or classical cytokines such as the pro-inflammatory mediators TNF-α and interleukin 6 (IL-6) (Hotamisligil, 2006; Tilg *et al.,* 2006).

On the other hand, reported studies have found an inverse relationship between lung function and markers of systemic inflammation (Amina *et al.,* 2010). So that, impaired respiratory function such as FEV1 is strongly related with cardiovascular risk factors, atherosclerosis, arterial stiffness, cardiovascular disease and mortality, although the mechanisms underlying this response are a matter of some debate. These changes are associated with disturbance in immune response in the lung. High sensitivity C-reactive protein is an inflammatory mediator known to be related to inflammation, and

cardiovascular diseases (Eizadi *et al.,* 2011). On the other hand, a positive relationship has been reported between raised CRP levels and current asthma (Ford, 2005; Jousilahti *et al.,* 2002), respiratory impairment (Danesh *et al.,* 2004; Gann *et al.,* 2004), and bronchial responsiveness (Mendall *et al.,* 2000).

Table 1. Mean and standard deviation of anthropometrical and biochemical variables in baseline and after intervention.

Variables	Control group		Exercise group	
	Pretest	post-test	Pretest	post-test
Age (years)	39 ± 5	39 ± 5	40 ± 4	40 ± 4
Weight (kg)	88 ± 9.6	89 ± 5.6	90 ± 11	86 ± 12
Height (cm)	174 ± 8.8	174 ± 8.8	175 ± 9.5	175 ± 9.5
Body fat (%)	27.4 ± 4.1	27.6 ± 3.8	28 ± 3.2	26 ± 3.11
Body mass index (kg/m²)	29.06 ± 2.9	29.36 ± 3.4	29.83 ± 2.6	28.08 ± 3.14
Visceral fat	11 ± 3.5	11.09 ± 2.9	11.3 ± 2.3	10 ± 1.2
Serum CEP (µg/ml)	2911 ± 512	2815 ± 341	2774 ± 568	2185 ± 418

Table 1. Mean and standard deviation of anthropometrical and biochemical variables in baseline and after intervention.

A recent study has been reported positive association between raised hs-CRP levels, current asthma, respiratory impairment and bronchial hyper-reactivity (Kony *et al.,* 2004; Jousilahti *et al.,* 2002). The relation between asthma and CRP is by no means clear, however, and could at least in part reflect the role of obesity in CRP production (Yudkin *et al.,* 1999). Some recent study showed that CRP level in stable asthma is significantly higher compared to those without asthma state. These researchers support the hypothesis that not only local but also systemic inflammation exists in bronchial asthma. To support this data, Jousilhti et al (Jousilahti *et al.,* 2002) demonstrated that asthma increased gradually with increasing CRP. The finding of another showed an inverse linear relationship between CRP concentrations and measures of pulmonary function in subjects without pulmonary disease and in never-smokers (Doron *et al.,* 2006). It was reported that exercise training for long time is associated with low systemic inflammation in chronic diseases (Tang *et al.,* 2005; Huang *et al.,* 2007), although the molecular mechanisms for this are less understood.

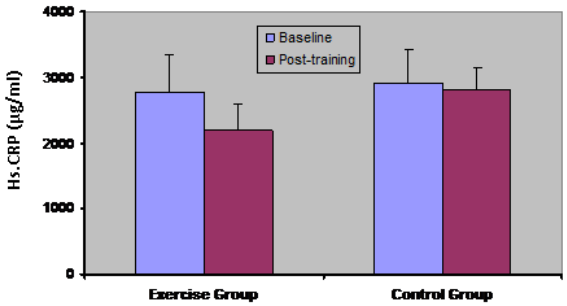

Fig. 1. The changes pattern of serum Hs.CRP in baseline and by interventions in two groups.

In present study, we observed a significant decrease in serum CRP by three months aerobic exercise program in asthma patients. On the other hand, decreased serum CRP was accompanied a reduction in all anthropometrical markers such as body weight, BMI, body fat percentage and visceral fat.

Since most previous studies have emphasized the fact that amplified adipose tissues especially abdominal obesity is associated with increased inflammatory cytokine, it appears reduced body weight along with decreased body in the subjects caused by exercise is one of the main reasons of decrease in serum levels of CRP. In fact, it appears that reduced levels of body fat through exercise may be attributed to reduced

secretion of this inflammatory cytokine from adipose tissue into the bloodstream. In this context, some other studies on obese subjects or subjects other chronic diseases such as diabetes or cardiovascular diseases have also reported that once physical activity is associated with a significant reduction in body fat levels it leads to the decrease of inflammatory cytokines, such as IL-6 - TNF-α and ... or the increase of anti-inflammatory cytokine (Varady *et al.,* 2009). Despite these findings, failure to measure other cytokines is one of the main limitations of this study which calls for evaluation in future studies.

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References

Amina H, Abdul G, Abdul K, Jasim M .2010. Association Between C Reactive Protein and Asthma. TurkishTorax Dergisi **11(3),** 098 -104.

Butland BK, Strachan DP, Rudnicka AR. 2008. C-reactive protein, obesity, atopy and asthma symptoms in middle-aged British adults. Eur Respir J **32,** 77-84.

Danesh J, Wheeler JG, Hirschfield GM. 2004. C–reactive protein and other circulating markers of inflammationin the prediction of coronary heart disease. N Eng J Med **350,** 387-97.

de Salles BF, Simão R, Fleck SJ, Dias I, Kraemer-Aguiar LG, Bouskela E. 2010. Effects of resistance training on cytokines. Int J Sports Med **31(7),** 441-50.

Doron A, Inon R, Mordechay Y, Arthur K, Ophir A, Ron S, Peter B. 2006. Inverse Association between Pulmonary Function and C - reactive protein in Apparently Healthy Subjects. Am J Respir Crit Care Med **174,** 626–632.

Ebrahim R , Hassan E, Hossein A, Vajihe C, Armin R. 2011. Armin Razi Evaluation of High-Sensitivity C-Reactive Protein in Acute Asthma Tanaffos **11(1),** 32-37.

Eizadi M, Kohandel M, kasbparast JR M, Sarshin A. 2011. Hs-CRP and TNF- alpha in response to a stepwise incremental bicycle test in adult obese men **1(3),** 35-43.

Ford ES. 2003. Asthma, body mass index, and C-reactive protein among US adults. Journal of Asthma **40(7),** 733- 9.

Ford ES. 2005. The epidemiology of obesity and asthma. J Allergy Clin Immunol **115,** 897-909.

Gann WQ, Man SFP, Senthilselvan A. 2004. Association between chronic obstructive pulmonary disease and systemic inflammation: a systematic review and meta analysis. Thorax **59,** 574-80.

Heidenfelder B, Johnson M, Hudgens E, Inmon J, Hamilton RG, Neas L, Gallagher JE. 2010. Increased plasma reactive oxidant levels and their relationship to blood cells, total IgE, and allergen-specific IgE levels in asthmatic children. J Asthma **47(1),** 106-11.

Hotamisligil GS. 2006. Inflammation and metabolic disorders. Nature **444,** 860-7.

Huang H, Iida KT, Sone H, Ajisaka R. 2007. The regulation of adiponectin receptors expression by acute exercise in mice. Exp Clin Endocrinol Diabetes **115(7),** 417-22.

Jousilahti P, Salomaa V, Hakala K, Rasi V, Vantera E, Palosuo T. 2002. The association of sensitive inflammation markers with bronchial asthma. Annals of Allergy, Asthma and Immunology **89(4),** 381-5.

Jousilahti P, Salomaa V, Hakala K, Rasi V, Vantera E, Palosuo T. 2002. The association of sensitive inflammation markers with bronchial asthma. Ann Allergy Asthma Immunol **89,** 381-5.

Jousilahti P, Salomaa V, Hakala K. 2002. The association of sensitive systemic inflammation markers with bronchial asthma. Ann Allergy Asthma Immunol **89,** 381-5.

Kony S, Zureik M, Driss F, Neukirch C, Leynaert B, Neukirch F. 2004. Association of bronchial hyperresponsiveness and lung function with C-reactive protein (CRP): a population based study. Thorax **59,** 892-6.

Kony S, Zureik M, Driss F. 2004. Association of BHR and lung function with CRP: a population based study. Thorax **59,** 1-5.

Mendall MA, Strachan DP, Butland BK. 2000. C-reactive protein: relation to total mortality, cardiovascular mortality and cardiovascular risk factors in men. Eur Heart J **21,** 1584-90.

Mendall MA, Strachan DP, Butland BK. 2000. C-reactive protein: relation to total mortality, cardiovascular mortality and cardiovascular risk factors in men. Eur Heart J **21,** 1584-90.

O'Connor GT, Sparrow D, Weiss ST. 1995. A prospective longitudinal study of methacholine airway responsiveness as a predictor of pulmonary-function decline: the Normative Aging Study. Am J Respir Crit Care Med 152, 87-92.

Olafsdottir IS, Gislason T, Thjodleifsson B, Olafsson I, Gislason D, Jögi R. 2005. C reactive protein levels are increased in nonallergic but not allergic asthma: a multicentre epidemiological study. Thorax **60(6),** 451- 4.

Ridker PM. 2000. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. N Engl J Med **342,** 836–843.

Rijcken BJ, Schouten JP, Xu X. 1995. Airway hyperresponsiveness to histamine associated with accelerated decline in FEV1. Am J Respir Crit Care Med **51,** 1377-82.

Sahoo RC, Acharya PR, Noushad TH, Anand R, Acharya VK, Sahu KR. 2009. A Study of High-Sensitivity C - reactive protein in Bronchial Asthma. Indian journal of chest diseases & allied sciences **51(4),** 213-6.

Tang Z, Yuan L, Gu C, Liu Y, Zhu L. 2005. Effect of exercise on the expression of adiponectin mRNA and GLUT4 mRNA in type 2 diabetic rats. J Huazhong Univ Sci Technolog Med Sci **25(2),** 191-3.

Tice JA. 2003. The relation of C-reactive protein levels to total and cardiovascular mortality in older U.S. women. Am J Med **114,** 199–205.

Tilg H, Moschen AR. 2006. Adipocytokines: mediators linking adipose tissue, inflammation and immunity. Nat Rev Immunol **6,** 772-83.

Varady KA, Tussing L, Bhutani S, Braunschweig CL. 2009. Degree of weight loss required to improve adipokine concentrations and decrease fat cell size in severely obese women. Metabolism 2009 **58(8),** 1096-101.

Yudkin JS, Stehouwer CD, Emeis JJ. 1999. C reactive protein in healthy subjects: associations with obesity, insulin resistance and endothelial dysfunction: a potential role for cytokines originating from adipose tissue? Arterioscler Thromb Vasc Biol. **19,** 972-8.