Biological Forum – An International Journal **7**(1): 365-370(2015)

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

Adipose Secreted Resistin In Response to Submaximal Exercise in Respiratory Patients

Jenabi Ahmad, Dastigerdi Mehdi and Abbasi Mohammadsaleh
Sama Technical and Vocational Training College,
Islamic Azad University, Andisheh Branch, Andisheh, IRAN

(Corresponding author: Jenabi Ahmad)
(Received 07 February, 2015, Accepted 21 February, 2015)
(Published by Research Trend, Website: www.researchtrend.net)

ABSTRACT: Obesity is considered to be a risk factor for asthma cardiovascular or respiratory diseases. This study aimed to determine the association of serum resistin as inflammatory adipokine with obesity determinants and its acute response to a submaximal exercise test in asthma patients. For this purpose, serum resistin and anthropometrical markers were measured in fourteen adult males with mild to moderate asthma. Serum resistin was also measured after a submaximal exercise test. Data were evaluated using, paired t and Pearson's tests. Serum resistin was positively associated with anthropometrical marked such as body weight, abdominal circumference, BMI and body fat (%) as obesity determinants ($p < 0.05$). serum resistin concentration did not change by exercise test when compared to pretest ($p = 0.65$). Our finding indicates that a submaximal exercise test that is not associated with negative energy balance has no significant acute effect on serum resistin in asthma patients.

Keywords: Submaximal exercise, Resistin, Inflammation, Respiratory diseases

INTRODUCTION

Over the past two decades, it has been repeatedly proposed that adipocytokines have a role in obesity and insulin resistance [1]. Most clinical studies support an increased adipokines or inflammatory adipocytokines or reduced anti-inflammatory cytokines in the presence of obesity [2, 3]. The role of theses peptide mediators has been frequently suggested in obesity-related diseases and other chronic diseases associated with metabolic disorders such as type 2 diabetes, atherosclerosis, and insulin resistance [1, 4].

Among the inflammatory mediators, the role of resistin in metabolic disorders has always been pointed out. Resistin is often produced in the adipose tissue. It reduces the insulin sensitivity in the adipose and skeletal muscle tissues through reducing the glucose transport [5]. It has an effective role in obesity, inflammation, atherosclerosis, and insulin resistance [6] as an increased resistin is associated with the increased occurrence of obesity and obesity-related diseases. However, some studies have reported no difference in this inflammatory mediator between healthy and diabetic subjects. The also reported the lack of correlation between resistin and body mass index as one of the indices of obesity in diabetic patients [7].

Some studies have reported that the role of resistin in the inflammatory disease is independent of the BMI levels [5]. Despite the observations regarding the role of resistin in obesity and obesity-related diseases such as type 2 diabetes, the literature on the importance of this inflammatory mediator in the pathophysiology of respiratory diseases, such as asthma or chronic obstructive pulmonary disease, are limited. However, some studies have reported obesity as an underlying factor in the occurrence or aggravation of asthma [8] as in one study, after adjustment for age, sex, level of daily activity, smoking and educational level, a close relationship was observed between asthma and obesity [9].

Scientific findings suggest that resistin has a predictive role in the occurrence of asthma [10], and its levels are significantly higher in asthmatic patients compared to healthy individuals [11]. The question is whether the serum resistin levels are associated with obesity indices in asthmatic patients. On the other hand, several studies have support the beneficial effects of exercise in improving the inflammatory profile in obese populations or patients with type 2 diabetes [12, 13]. Researchers attribute the improved inflammatory profile, such as resistin, to the aerobic training-induced weight loss in these subjects [14, 15].

Some studies have also reported the unchanged inflammatory profile in response to exercise-induced weight loss [16]. However, the effects of the short-term exercise tests on inflammatory or anti-inflammatory cytokines, particularly in asthmatic patients, have not been addressed much. Hence, in the present study, the relationship between serum resistin and obesity indices in asthmatic patients was determined, and the effect of a submaximal exercise test on this inflammatory mediator was evaluated.

MATERIALS AND METHODS

A. Subjects

Fourteen untrained, nonsmoking adult males with asthma aged 35 - 50 years and BMI: 26–36 kg/m2 were recruited for participate in this study. Based on Spirometrical parameters, asthma severity was mild to moderate. The study was conducted with the approval of the Ethics Committee of Islamic Azad University, Iran and an informed consent was obtained from all participants before the studies were carried out. Those with a self reported physician diagnosed chronic disease such as type II diabetes, arthritis, stroke, hypertension, heart attack and other chronic diseases were excluded. All participants had not participated in regular exercise/diet programs for the preceding 6 months.

B. Anthropometrics and spirometry

Each subject's anthropometrical markers were measured. Body weight and height were measured on the same day to the nearest 0.1 kg and the nearest 0.1 cm, respectively. BMI was calculated as weight (kilograms) divided by height squared (square meters). Waist circumference (WC) was measured at the superior border of the iliac crest and was taken to the nearest 0.1 cm after a normal expiration, while the hip girth was measured at the level of the greatest protrusion of the gluteal muscles with underwear. Each of these measurements was conducted three times and the average was reported.

Subjects were asked to refrain from tea, coffee, chocolates and caffeinated soft-drinks on the day of recording Spirometry. Subjects were instructed to take maximum inspiration and blow into the pre-vent pneumotach as rapidly, forcefully and completely as possible for a minimum of 6 seconds, followed by full and rapid inspiration to complete the flow volume loop. The best of the three trials was considered for data analysis.

C. Blood analysis and submaximal protocol

Venous blood samples were obtained before and immediately after exercise test for measure serum resistin by ELIZA method. The inter- and intra-assay coefficients of variance were 3.4 and 6.9 % for resistin. All subjects underwent a submaximal exercise test included running at 70 (%) of maximal heart rate for 40 min. exercise intensity was equal for all participants. Participants wore heart rate monitors to ensure that they were reaching target heart rate levels.

D. Data analysis

Data were analyzed by computer using the Statistical Package for Social Sciences (SPSS) for Windows, version 15.0. Normality of distribution was assessed by Kolmogorov-Smirnov test. Pearson correlation was used to determine the association between serum resistin. Student's t-tests for paired samples were performed to determine whether there was significant difference in serum resistin between pre and post test. A p-value less than 0.05 were considered statistically significant.

RESULTS

In present study, the relation of serum resistin with anthropometrical markers were determined in asthma patients at first.

The physical characteristics and Spirometrical markers of the subjects are shown in Table 1. Based on person correlation coefficients, a significant correlation was found in serum resistin and body weight in studied patients ($p = 0.002$, $r = 0.71$, Fig. 1). Serum resistin was also positively correlated with abdominal circumference ($p = 0.005$, $r = 0.66$, Fig. 2).

Pearson analysis methods revealed that BMI ($p = 0.001$, $r = 0.73$, Fig. 3) and body fat percentage ($p = 0.026$, $r = 0.59$, Fig. 4) were positively associated with serum resistin levels.

Acute response of serum resistin to submaximal exercise test was also main objective of our study. Based on Paired samples T test, there was no statistically significant difference in serum resistin between pre and post exercise (from 7.78 ± 2.42 to 8.11 ± 3.25 ng/ml, $p = 0.649$, Fig. 5). On the other hand, submaximal exercise test was not associated with anti-inflammatory property based on serum resistin in asthma patients.

Table 1: The descriptive anthropometric and biochemical features of the study subjects.

Variables	Minimum	Maximum	Mean	Std. Deviation
Age (year)	37	55	44.31	5.43
Height (cm)	172	179	175.2	2.04
Weight (kg)	79	118	92.7	11.35
Abdominal (cm)	94	132	105.9	9.53
Hip (cm)	92	126	104.3	8.49
WHO	.96	1.11	1.02	0.04
BMI (kg/m2)	26.4	36.80	30.16	3.31
Body fat (%)	17.3	38.1	29.3	4.70
FVC (%)	73	92	82.2	5.82
FEV1 (%)	65	86	77.7	6.29
FEV1 / FVC (%)	64	70	66.9	1.746
Resistin (ng/ml, pre-test)	4.3	12.3	7.88	2.42
Resistin (ng/ml, post-test)	3.5	17.6	8.1	3.25

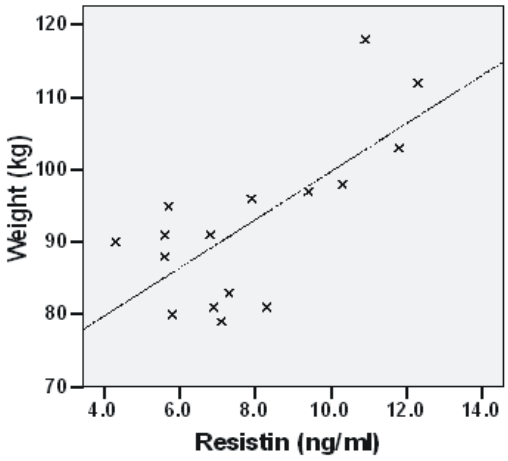

Fig. 1. Positive correlation of serum resistin (cm) in asthma patients.

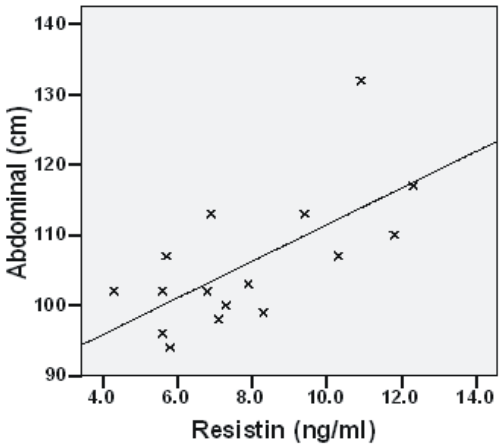

Fig. 2. Positive correlation of serum resistin with abdominal with body weight in asthma patients.

DISCUSSION

The findings showed a significant relationship between serum resistin and anthropometric indices in asthmatic patients. Consistent with previous findings, this study also showed a significant relationship between serum resistin levels and weight, waist circumference, and percent body fat in patients with asthma. The relationship between serum resistin levels with obesity indices in asthmatic patients somehow supports the role of obesity in resistin levels in asthmatic patients and somehow indicates the presence of inflammation in obese subjects.

The available evidence suggests that the relationship between serum resistin levels and obesity indices are affected by the presence of disease. According to a study, despite the close correlation between serum resistin levels and weight, hip circumference, abdominal circumference and fasting glucose in patients with type 2 diabetes, no correlation was observed in healthy individuals [4]. However, the lack of correlation between serum resistin and BMI in diabetic patients has also been reported [7]. Some studies have also reported a relationship between resistin and inflammatory symptoms independent of BMI [17].

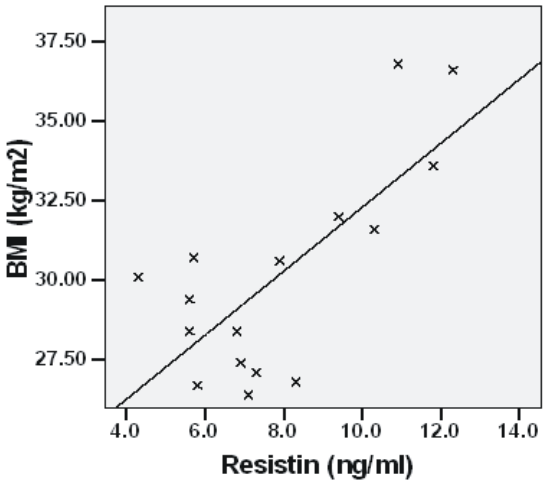

Fig. 3. Positive correlation of serum resistin with BMI in asthma patients.

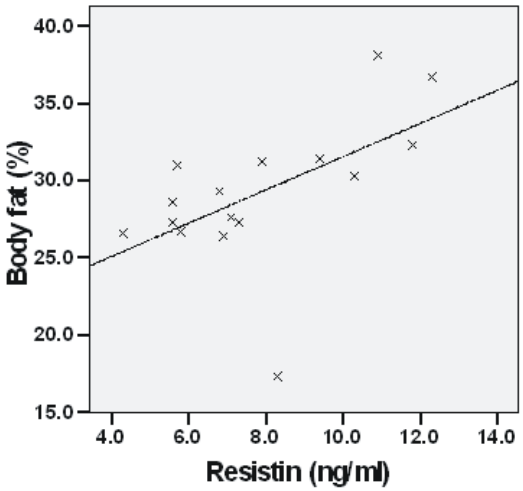

Fig. 4. Positive correlation of serum resistin with body fat (%) in asthma patients.

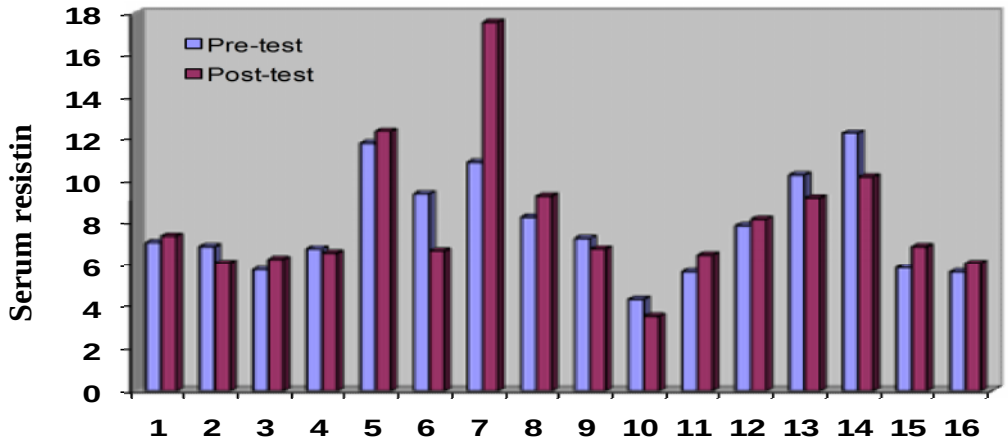

Fig. 5. Pre and post exercise of serum resistin of studied subjects. Each number of horizontal axis represent one subjects. Serum resistin did not change by exercise test in all subjects.

Secretion of some adipocytokine, such as leptin, otaxin, resistin, and inflammatory mediators such as TNF-a, CRP, and IL-6, IL-1b, and reduced anti-inflammatory adipokines such adiponectin in the obese individuals plays a key role in the occurrence or the aggravation of asthma or chronic obstructive pulmonary disease clinical symptoms and inflammation of airways [18]. Although the exact mechanism of the association between asthma and obesity is not fully understood yet, some studies have shown a causal relationship between them [19]. It seems that damaged lung function and mechanism induced by obesity lead to an increased resistance of airways. In other words, asthma is an inflammatory disease, and research findings note obesity as a factor increasing inflammation symptoms [20]. Studies suggest the extensive role of obesity on the occurrence of asthma and reduced quality of life [8]. The findings indicate that after adjustment for tobacco use and the physical activity and educational level, the frequency of asthmatic patients are higher in obese or overweight subjects compared to healthy individuals [9].

Sustained weight loss in obese asthmatic patients is associated with the improved respiratory symptoms, and improved lung and nervous system functions [21]. Studies suggest that obesity precipitates the onset of asthma. They believe that an increased obesity increases the severity of asthma, and weight loss improves and reduces the severity of asthma [22]. Weight loss in obese individuals reduces asthma symptoms [23). Hence, similar to other diseases, it seems that exercise-induced weight loss in patients with asthma is associated with improved inflammatory profile. However, findings regarding the role of short-term exercise tests on the levels of these cytokines, especially serum resistin, in patients with asthma are limited. In the present study, a submaximal aerobic training did not changes the levels of serum resistin in patients with asthma. In other words, 40 minutes of moderate-intensity running did not affect the resistin levels in these patients. In support of these findings, some other studies also reported the unchanged levels of inflammatory mediators in response to single-session exercise tests in other healthy or patient populations [24].

However, there are studies that support the reduced inflammatory cytokine or increased anti-inflammatory cytokines in response to a single exercise session [25]. The inconsistency in findings may be due to differences in the type, intensity, and duration of exercise, time of blood sampling, or differences in the studied populations. However, some studies that have examined this issue more rigorously have noted that only those exercises that lead to the negative energy balance or had am energy cost of 80 kcal improve the inflammatory profile [26, 27]. Based on these statements, the lack of reduction in serum resistin levels in asthmatic patients in response to a 40-minute exercise test is not unexpected in the present study.

REFERENCES

Mojiminiyi OA, Abdella NA. (2007). Associations of resistin with inflammation and insulin resistance in patients with type 2 diabetes mellitus. *Scand J Clin Lab Invest.* **67**(2): 215-25.

Aydin M, Koca C, Ozol D, Uysal S, Yildirim Z, Kavakli HS, Yigitoglu MR. (2013). Interaction of metabolic syndrome with asthma in postmenopausal women: role of adipokines. *Inflammation.* **36**(6): 1232-8.

Huang CJ, Zourdos MC, Jo E, Ormsbee MJ. (2013). Influence of physical activity and nutrition on obesity-related immune function. *Scientific World Journal.* **3**: 752071.

Al-Harithy RN, Al-Ghamdi S. (2005). Serum resistin, adiposity and insulin resistance in Saudi women with type 2 diabetes mellitus. *Ann Saudi Med.* **25**(4):283-7.

Palanivel R, Maida A, Liu Y, Sweeney G. (2006). Regulation of insulin signalling, glucose uptake and metabolism in rat skeletal muscle cells upon prolonged exposure to resistin. *Diabetologia.* **49**: 183-190.

Steppan CM, Bailey ST, Bhat S. (2001). The hormone resistin links obesity to diabetes. *Nature.* **409**: 307–312.

Stejskal D, Adamovská S, Bartek J, Juráková R, Prosková J. (2003). Resistin - concentrations in persons with type 2 diabetes mellitus and in individuals with acute inflammatory disease. *Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub.* **147**(1): 63-9.

Hersoug LG, Linneberg A. (2007). The link between the epidemics of obesity and allergic diseases: does obesity induce decreased immune tolerance? *Allergy*. **62**(10): 1205-13.

Castro-Rodríguez JA. (2007). Relationship between obesity and asthma. *Arch Bronconeumol.* **43**(3):171-5.

Kim KW, Shin YH, Lee KE, Kim ES, Sohn MH, Kim KE. (2008). Relationship between adipokines and manifestations of childhood asthma. *Pediatr Allergy Immunol.* [Epub ahead of print].

Larochelle J, Freiler J, Dice J, Hagan L. (2007). Plasma resistin levels in asthmatics as a marker of disease state. *J Asthma*. **44**(7): 509-13.

Wei SS, Liang DD. Zhongguo Ying Yong Sheng Li Xue Za Zhi. (2013). The effect of exercises on TNF-alpha, IL-6 and adiponectin in different fat diet rats. **29(**3): 280-2.

Ko IG, Choi PB. R. (2013). Regular exercise modulates obesity factors and body composition in sturdy men. *J Exerc Rehabil*. **9**(2): 256-62.

Park S, Han T, Ann E, Yoon E, Kim B. (2008). Effect of training intensity on subcutaneous adipose leptin, adiponectin, interleukin 6, and tumor necrosis factor mRNA expression in middle-aged women". *FASEB J*. **22**: 118.

Jung SH, Park HS, Kim KS, Choi WH, Ahn CW, Kim BT, Kim SM, Lee SY, Ahn SM, Kim YK. (2008). Effect of weight loss on some serum cytokines in human obesity: increase in IL-10 after weight loss. *J Nutr Biochem.* **19**(6): 371-5.

de Luis DA, Aller R, Izaola O, Gonzalez Sagrado M, Bellioo D, Conde R. (2007). Effects of a low-fat versus a low-carbohydrate diet on adipocytokines in obese adults. *Horm Res.* **67**(6): 296-300.

Shetty GK, Economides PA, Horton ES, Mantzoros CS, Veves A. (2004). Circulating adiponectin and resistin levels in relation to metabolic factors, inflammatory markers, and vascular reactivity in diabetic patients and subjects at risk for diabetes. *Diabetes Care*. **27**(10): 2450-7.

Boulet LP. (2008). Influence of obesity on the prevalence and clinical features of asthma. *Clin Invest Med.* **31**(6): 386-90.

Hancox RJ, Milne BJ, Poulton R, Taylor Dr, Greene JM, Mclachlan CR et al. (2005). Sex differences in the relation between body max index and asthma and atopy in a birth cohort. *Am J Respir Crit Care Med.* **171**: 440-5.

Visser M, Bouter LM, McQuillian GM, Wener MH, Harris TB. (2001). Low-grade systemic inflammation in overweight children. *Pediatrics.* **107**: 13.

Shore SA. (2007). Obesity and asthma: implications for treatment. C*urr Opin Pulm Med.* **13**(1): 56-62.

Jung SH, Park HS, Kim KS, Choi WH, Ahn CW, Kim BT. (2008). Effect of weight loss on some serum cytokines in human obesity: increase in IL-10 after weight loss. *J Nutr Biochem.* **19**(6): 371-5.

Valsamakis G, McTernan PG, Chetty R, Al Daghri N, Field A, Hanif W. (2004). Modest weight loss and reduction in waist circumference after medical treatment are associated with favorable changes in serum adipocytokines. *Metabolism.* **53**(4): 430-4.

Eizadi M, Dooaly H, Kiani F, Khorshidi F. (2013). Effects of cycling test on serum interleukin 6 and lipid profile in asthma patients. *International Journal of Biosciences.* **3**(2): 158-162.

Zwetsloot KA, John CS, Lawrence MM, Battista RA, Shanely RA.(2014). High-intensity interval training induces a modest systemic inflammatory response in active, young men. *J Inflamm Res.* **7**: 9-17.

Kraemer RR, Chu H, Castracane DV. (2000). Leptin and exercise. *Exp. Biol. Med.* **227**: 701–708.

Hqjbjerref L, Rozenzweig M, Dela F, Bruun JM, Stallknecht B. (2007). Acute exercise increases adipose tissue interstitial adiponectin concentration in healthy overweight and lean subjects. *European Journal of Endocrinology.* **157**(5): 613–623.