



Effect of a Chronic Aerobic Training on Adipocytokine Resistin in Smokers

Hajirasouli Masoud, Hajirasouli Maral and Afsharmand Zohreh
Department of Physical Education and Sport Sciences,
Islamshahr Branch, Islamic Azad University, Islamshahr, IRAN

(Corresponding author: Hajirasouli Masoud)

(Received 26 September, 2014, Accepted 28 October, 2014)

(Published by Research Trend, Website: www.researchtrend.net)

ABSTRACT: Previous epidemiological studies support an important contribution of smoking to the development of insulin resistance and cardiovascular diseases. In present study, we aimed to evaluate effect of aerobic training on serum resistin in smokers, a totally twenty six sedentary male smokers were randomly selected into experimental and control groups. Anthropometrical and fasting serum resistin were measured before and after 10 weeks aerobic training in experimental as well as control (no training) subjects. Comparisons of the study variables were performed between groups by one-way analysis of variance (ANOVA). No differences in resistin concentration and anthropometrical markers were found between two groups at baseline ($p > 0.05$). Significant decrease was observed after training program compared to their respective pre test values of anthropometrical markers in experimental groups ($p < 0.05$). Serum resistin was also increased by training program in experimental subjects ($p = 0.012$). Increased serum resistin by training program could be attributed to high exercise intensity or cigarette smoking during exercise intervention by smokers.

Keywords: Smoke, Body weight, Aerobic training, Sedentary

INTRODUCTION

In addition to numerous mental and psychological abnormalities, problems such as various kinds of cardiovascular diseases, type 2 diabetes, respiratory and kidney diseases, and some types of cancer are among the serious complications resulting from smoking not only in smokers but also in those who are around them. The relationship between smoking and systemic inflammation and cardiovascular diseases has been reported numerous times [1], and release of inflammation mediators caused by smoking has been suggested as the factor preparing the ground for the prevalence of many chronic and inflammatory diseases [2, 3].

Some complications resulting from smoking are reversible, but the harmful effects of other complications such as suppression of immune responses by inflammation intermediaries are so severe that they persist years after smokers quit [4]. Among the adipocytokines, resistin is a newly discovered hormone that is secreted by adipose tissue and plays an effective role in the relationship between obesity and insulin resistance [5]. Preliminary studies have shown resistin levels rise in obese and diabetic people [5]. In this relation, Menzaghi (2013), based on findings in her

research, supported the opinion that high resistin levels were a risk factor for cardiovascular and other diseases related to diabetes [6]. Moreover, it has been proved that plasma-resistin levels are of special importance in the prevalence and severity of gestational diabetes because of their role in insulin sensitivity and resistance [7, 8]. Researchers have also supported reports that resistin levels rise in smokers [9], and it has been reported that there is a negative relationship between resistin and spirometric indices in smokers [10], which somewhat supports the idea that resistin plays a role in respiratory function and airway resistance in smokers and in other respiratory diseases such as asthma [10]. Nevertheless, some scientists have reported resistin is not related to other inflammatory markers such as CRP and homocysteine in smokers [11].

Despite the fact that a considerable number of studies have been carried out, limited research has been conducted on resistin response to various training programs in smokers. Moreover, scientific findings in other healthy or sick populations are contradictory to some extent so that those of a recent study showed a significant reduction in serum resistin levels following 12 months of aerobic training by obese people [12].

However, in another study, concentration of this intermediary peptide did not significantly change after 14 months of training [13]. Some other studies have also observed significant reductions in serum resistin levels in the absence of significant decreases in body weight [14]. Based on contradictory observations regarding resistin response to training programs in other populations, and since limited research has been carried out on smokers, this study was conducted to determine what effects 10 weeks of aerobic training had on serum resistin levels in male smokers.

MATERIALS AND METHODS

Twenty six sedentary male smokers matched for age (35-45, aged) were randomly assigned to either a 10 weeks aerobic training group (experimental, $n = 13$) or a control group (no training, $n = 13$). The study protocol was approved by the ethics committee of Islamic Azad University and written informed consent was obtained from all participants. Subjects were excluded if they had not a chronic disease and able to participate in exercise training. A smoker is defined as a regular cigarette at least for 3 years. All subjects had not participated in regular exercise/diet programs for the preceding 6 months.

A. Anthropometry

Anthropometric measurements (body height and weight, waist and hip circumference) were performed with the subjects wearing light underwear and without shoes. Body weight and height were measured on the same day to the nearest 0.1 kg and the nearest 0.1 cm, respectively. Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter. BMI was calculated as weight (kg)/height (m²). Blood pressure (BP) was measured with a mercury tensiometer at the right arm, with elbow flexed at heart level with the subject in a seated position after 5 minutes of rest. Two measurements were made every 1 minute and the average of two measurements was used for analysis.

B. Blood samples and training program

All anthropometrical measurements were repeated after training program. Blood samples were also collected before and after (48 hours) intervention. Blood was obtained after a overnight fast between the hours of 8 to 9 am and used to serum resistin. Serum was immediately separated and stored at 70 °C until biochemical analyses were performed.

Exercise program lasted 10 weeks and subjects asked to participate in a set of exercises 3 days a week that supervised by a trained exercise physiologist. Exercise session involved a warm-up then a 45-60 min aerobic training at a work intensity of 60-80% of peak heart rate followed by a cooling-down period. Main exercise in each session was walking on running at mentioned intensity. The exercise intensity at first week was 60 % of Max heart rate that gradually increased at lasted sessions of exercise program.

C. Statistical analysis

All statistical analyses were performed through the use of a statistical software package (SPSS, Version 15.0, SPSS Inc., IL, USA). The Kolmogorov-Smirnov test was applied to determine the variables with normal distribution. One way analysis of variance was used to determine whether the changes in the outcomes were significantly different between groups. A criterion alpha level of $P = 0.05$ was used for all statistical comparisons.

RESULTS

Our study objective was to assess effects of ten weeks aerobic training on serum resistin in males smokers. This study involved experimental and control subjects.

At baseline, there were no significant differences in anthropometrical markers between two groups ($p = 0.05$). No difference was also observed between two groups with regard to serum resistin at baseline ($p = 0.764$).

Anthropometrical and blood chemistry parameters during experimental protocol of control groups are shown in Table 1. Anthropometrical markers were not significantly changed after 10 weeks no training in control subjects ($p = 0.05$).

Table 2 shows the changes in body weight, other anthropometrical markers and serum resistin of experimental group. Based on data of statistical analysis, all anthropometrical markers such as body weight, body mass index, body fat percentage, abdominal circumference decreased significantly by aerobic training in experimental subjects when compared with their pretest values ($p < 0.05$).

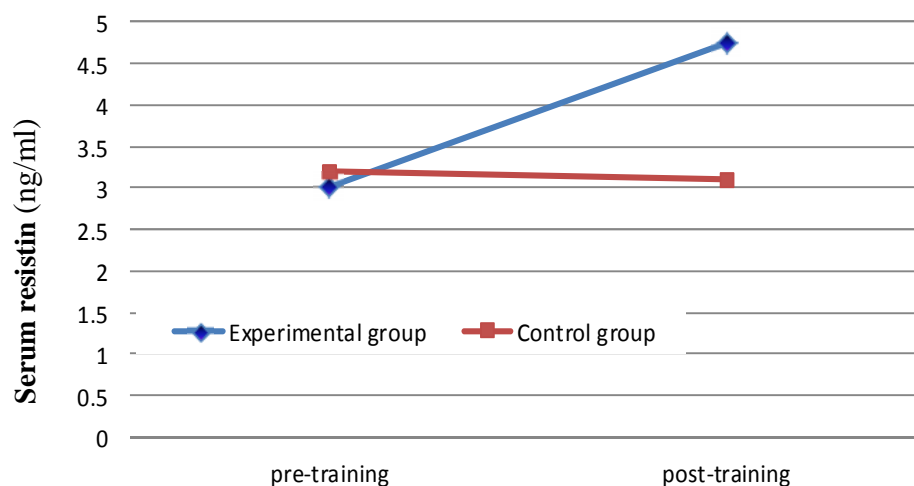
Aerobic training was also resulted in significant increase in serum resistin in experimental group (from 3.02 ± 1.89 ng/ml to 4.74 ± 2.04 ng/ml, $p = 0.042$) but this adipocytokine remained unchanged in control subjects ($p = 0.793$) (Fig. 1).

Table 1: The descriptive anthropometric markers and resistin before and after training program of experimental group.

| Variables | Pre-training | Post-training |
|--------------------------------------|--------------|---------------|
| Age (year) | 41.2 ± 6.8 | ----- |
| Height (cm) | 175.8 ± 3 | ----- |
| Systolic pressure (mmHG) | 132 ± 9 | 125 ± 8 |
| Diastolic pressure (mmHG) | 88 ± 8 | 86 ± 7 |
| Weight (kg) | 92.2 ± 5.26 | 88.2 ± 5.33 |
| Abdominal circumference (cm) | 101.8 ± 7.1 | 96.9 ± 6 |
| Hip circumference (cm) | 102 ± 5.4 | 96.8 ± 4.8 |
| Body mass index (kg/m ²) | 30 ± 2.14 | 28.6 ± 2.14 |
| Body Fat (%) | 28.9 ± 2.25 | 27.1 ± 1.74 |
| Resistin (ng/ml) | 3.02 ± 1.89 | 4.74 ± 2.04 |

Table 2: The descriptive anthropometric markers and resistin before and after intervention (no-training) of control group.

| Variables | Pre-test | Post-test |
|--------------------------------------|-------------|-------------|
| Age (year) | 10.8 ± 3.56 | ----- |
| Height (cm) | 175.4 ± 2.1 | ----- |
| Systolic pressure (mmHG) | 134 ± 14 | 130 ± 9 |
| Diastolic pressure (mmHG) | 90 ± 7 | 88 ± 8 |
| Weight (kg) | 90.9 ± 5.91 | 89.7 ± 5.35 |
| Abdominal circumference (cm) | 100 ± 5.2 | 99 ± 4.8 |
| Hip circumference (cm) | 100 ± 5.6 | 99 ± 5.8 |
| Body mass index (kg/m ²) | 29.5 ± 1.76 | 29.2 ± 1.68 |
| Body Fat (%) | 28.7 ± 1.6 | 28.5 ± 1.7 |
| Resistin (ng/ml) | 3.2 ± 1.1 | 3.1 ± 1.3 |

**Fig. 1:** Serum resistin before and after intervention in two groups. Aerobic program resulted in significant increase in serum resistin in experimental group.

DISCUSSION

Ten weeks of aerobic training significantly increased serum-resistin levels in male smokers and led to significant reductions in body weight. As was previously mentioned, some complications resulting from smoking persist even years after smokers quit. In this regard, findings of a study showed that some inflammation intermediaries such as CRP remained at high levels even 10 to 20 years after smokers quit, although some changes resulting from smoking were reversible after smokers quit [7].

Contrary to rodents, resistin in humans is expressed in large quantities in macrophages and its expression increases in response to various proinflammatory stimulants such as lipopolysaccharides, TNF- α , IL-6, IL-1 α , and resistin itself, which points to the role resistin plays in inflammation in humans [15, 16, 17].

Since there is a direct relationship between resistin levels and obesity, and also because studies have pointed to higher resistin levels in obese people compared to those with normal body weights [5, 18], it is expected reductions in body weight will be accompanied by reduced levels of resistin or other inflammation intermediaries. However, in this study, serum resistin significantly increased in response to the training program while there were significant reductions in body weight and body fat percentage. In this regard, some recent research has reported only those training programs influence levels of inflammatory cytokines that are accompanied by significant reductions in body weight [19], while it has been reported in other studies that levels of these cytokines do not change even when body weight decreases following long training programs [20].

Among recent observations, a study that was conducted recently found serum-resistin levels significantly decreased following 12 weeks of aerobic training by obese people [12], while in another study serum levels of this peptide intermediary did not significantly change after 14 weeks of training [13]. Some other studies have also reported significant reductions in resistin levels without any significant decreases in weight [14], but still in other studies serum-resistin levels significantly declined following long-term aerobic training while levels of other cytokines such as adiponectin and leptin [21] and CRP [22] did not change. Changes in serum resistin levels of smokers in response to training programs may also be independent of changes in body weight, as was pointed out in a recent study that found serum-resistin levels were independent of BMI [11].

Considering the existing contradictions, there is yet no consensus regarding the effects of aerobic training,

especially long term aerobic training, on levels of these cytokines in various populations, and a general summing up of this point seems to be somewhat difficult. Nevertheless, the main mechanisms responsible for the relationship between resistin and smoking have not been found yet. Increased resistin levels following training programs may be attributed to the fact that people participating in these intensive programs used to lead inactive lifestyles before the programs started. Moreover, this study was carried out just to determine the effects of the training programs on serum resistin levels, because the participants did quit smoking. Therefore, the rise in resistin levels may have been due to the combination of intensive training programs and smoking during the duration of the programs. Another possible mechanism for the increased resistin levels in response to the training programs may be attributed to the role this hormone plays in the antioxidant defense systems in the human body, because this hypothesis was put forward that resistin responded as an antioxidant to inflammatory stimulants [23]. These increases in serum levels of resistin in response to training programs were also attributed to this possible mechanism in the study Rashid Lemir *et al.* conducted in 2011.

REFERENCES

- Virginia R, Xiangying X, Diane B, Daniel J, Christine F, Patricia F *et al.* (2011). A Pilot Study To Examine the Effects of Smoking Cessation on Serum Markers of Inflammation in Women at Risk for Cardiovascular Disease. *Chest*. **3**: 212-219.
- 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-1 β 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, Creutzberg, Geertjan Wesseling and Emiel F. M. Wouters. (2007). Systemic Effects of Smoking. *CcChest*. **3**: 1557-1566.
- Steppan CM, Bailey ST, Bhat S, Brown EJ, Banerjee RR, Wright CM, Patel HR, Ahima RS, Lazar MA. (2001). The hormone resistin links obesity to diabetes. *Nature*. **409**:307-12.

- Menzaghi C, Bacci S, Salvemini L, Mendonca C, Palladino G, Fontana A, De Bonis C, Marucci A, Goheen E, Prudente S, Morini E, Rizza S, Kanagaki A, Fini G, Mangiacotti D, Federici M, De Cosmo S, Pellegrini F, Doria A, Trischitta V. (2013). Serum resistin, cardiovascular disease and all-cause mortality in patients with type 2 diabetes. *PLoS One*. **8**(6): 64729.
- Lobo TF, Torloni MR, Gueuvoghlian-Silva BY, Mattar R, Daher S. R(2013). Resistin concentration and gestational diabetes: a systematic review of the literature. *J Reprod Immunol*. **97**(1): 120-7.
- Eizadi M, Bananaeifar A, Khorshidi D, Dooaly H. (2011). Human serum resistin is related to homeostasis model assessment of insulin resistance in healthy people. *Journal of Biodiversity and Environmental Sciences*. **1**(4): 55-60.
- Gürsoy G, E bah O, Kirnap NG, Cetiner H, Acar Y, Demirba B, Oztürk A, Kiliç Z. (2012). The relation of obesity with serum resistin levels in smoker and nonsmokers. *J Res Med Sci*. **17**(2):119-22.
- Al Mutairi SS, Mojiminiyi OA, Shihab-Eldeen A, Al Rammah T, Abdella N. (2011). Putative roles of circulating resistin in patients with asthma, COPD and cigarette smokers. *Dis Markers*. **31**(1):1-7.
- Esbah O, Gürsoy G, Kirnap NG, Cetiner H, Demirba B, Acar Y, Bayram M. (2011). Relation of resistin levels with C-reactive protein, homocysteine and uric acid in smokers and non-smokers. *J Res Med Sci*. **16**(10): 1273-9.
- 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.
- Giannopoulou I, Fernhall B, Carhart R, Weinstock R, Baynard T, Figueroa A. (2005). Effects of diet and/or exercise on the adipocytokine and inflammatory cytokine levels of postmenopausal women with type 2 diabetes. *Metabolism*. **54**: 866-875.
- Kadoglou NP, Perrea D, Iliadis F, Angelopoulou N, Liapis C, Alevizos M. (2007). Exercise Reduces Resistin and Inflammatory Cytokines in Patients With Type 2 Diabetes. *Diabetes Care*. **30**(3): 719-21.
- Lehrke M, Reilly MP, Millington SC, Iqbal N, Rader DJ, Lazar MA. (2004). An inflammatory cascade leading to hyperresistinemia in humans. *PLoS Med*. **1**: 45.
- Kaser S, Kaser A, Sandhofer A, Ebenbichler CF, Tilg H, Patsch JR. (2003). Resistin messenger-RNA expression is increased by proinflammatory cytokines in vitro. *Biochem Biophys Res Commun*. **309**: 286-90.
- Bokarewa M, Nagaev I, Dahlberg L, Smith U, Tarkowski A. Resistin, an adipokine with potent proinflammatory properties. *J Immunol*. **174**: 5789-95.
- Patel L, Buckels AC, Kinghorn IJ, Murdock PR, Holbrook JD, Plumpton C, Macphee CH, Smith SA. (2005). Resistin is expressed in human macrophages and directly regulated by PPAR gamma activators. *Biochem Biophys Res Commun*. **300**: 472-6.
- 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.
- Wenning P, Kreutz T, Schmidt A, Opitz D, Graf C, Voss S, Bloch W, Brixius K.(2013). Endurance exercise alters cellular immune status and resistin concentrations in men suffering from non-insulin-dependent type 2 diabetes. *Exp Clin Endocrinol Diabetes*. **121**(8): 475-82.
- Many G, Hurtado ME, Tanner C, Houmar J, Gordish-Dressman H, Park JJ, Uwaifo G, Kraus W, Hagberg J, Hoffman E.(2013). Moderate-intensity aerobic training program improves insulin sensitivity and inflammatory markers in a pilot study of morbidly obese minority teens. *Pediatr Exerc Sci*. **25**(1):12-26.
- Bo S, Gambino R, Pagani A, Guidi S, Gentile L, Cassader M. (2005). Relationships between human serum resistin, inflammatory markers and insulin resistance. *Inter J Obese*. **29**(11): 1315-20.
- Rashidlamir A, Saadatnia A. (2011). Effects of 2 months aerobic training on glucose homeostasis index and cardiovascular risk factors. *J Shahid Sadoughi Univ Med Sci*. **19**(2): 219-29.