



Adipokine Resistin does not affect by Short Term Moderate Intensity Program in Obese Women

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ABSTRACT: Obesity has been related with metabolic abnormality and metabolic syndrome. The effect of short term aerobic training on serum resistin as inflammatory adipocytokine was investigated in obese female in present study. For this purpose, twenty four sedentary healthy obese females were participated in present study and divided into exercise (6 weeks aerobic training) or control groups by randomly. Pre and post training of serum resistin and anthropometrical markers were measured of two groups. Data was analyses by one way analysis variance. Serum resistin was positively correlated with abdominal obesity in studied subject at baseline ($p = 0.039$, $r = 0.42$) in studied subjects before exercise intervention. Despite improve in body weight and abdominal obesity, serum resistin concentration did not change significantly by training program compared to pre test ($p = 0.931$). Based on our finding and results of some previous studies, it seems that a weight loses at least 5 percent is necessary to improve adipocytokines in obese or related diseases.

Keywords: Adipocytokine, obesity, Exercise, Inflammation

INTRODUCTION

World Health Organization considers the rapid increase in the prevalence of obesity as an epidemic and identifies obesity and its complications as one of the major health problems in the world (1) So that diseases such as hyperglycemia, hypertension, atherosclerosis and heart disease are largely a consequence of obesity (2, 3).

It is now obvious that adipose tissue releases biologically active proteins, called Adipokines, spreading systemic inflammation in obese patients and associated diseases. Moreover, there is some evidence that adipocytes secrete a variety of protein signals such as inflammatory cytokines. The expression of various inflammatory genes, secreted from adipose tissue, is increased during obesity (4).

Among inflammatory mediators, it is a Resistin hormone secreted by adipose tissue that plays an important role in the regulation of energy homeostasis and metabolism. This inflammatory Adipokine with molecular weight of 12.5 kDa was discovered in 2001 (5). It secretes in adipocytes as well as muscle, pancreatic islet, human placenta and mononucleosis cells (6). It is of little expression in human adipose tissue, but it has more expression in the bone marrow, spleen, and placenta and lung tissue (6). Scientific resources supported the idea that Resistin levels are

increased in the presence of obesity, indicating its direct relation with insulin resistance in obese people (7) and its role in metabolic disorders and chronic diseases caused by obesity (5, 8).

Hence, creating appropriate environmental solutions such as changing the patterns of dietary behaviors has been health science researchers' primary goal to balance the levels of the peptide mediators in these individuals. On the other hand, as physical inactivity disrupts the systemic levels of this cytokines, obese people are often inactive, having a sedentary lifestyle. It is always thought that doing physical activities, whether short or long, and the resulted weight loss are somehow related to improvements of the cytokine levels in the obese patients and associated diseases. The findings are often contradictory in this regard, so that some studies have reported reduction (9) and some others have reported no change (10) of resistin and other Adipocytokines in response to a variety of training programs. Although most studies have attributed the contradictions in the findings to the differences in body weight changes, they considered the factors such as the type and intensity of the training program, gauges and the studied population to be effective in discrepancies between the findings. Based on these inconsistencies, the present study aimed to assess the effect of 6-week aerobic exercise on serum resistin levels in obese women.

MATERIALS AND METHODS

A. Human subjects and inclusion

Subjects were twenty four non-trained healthy obese matched for age, 30-45 years, height 155-170 cm and BMI 30-36 kg/m² that participated in this study by accessible samples. Subjects then divided into exercise or control groups by randomly. All participants of exercise group underwent a 6 weeks aerobic training program and control subject continued their daily activity.

The ethics approval was taken from Islamic Azad University of Iran ethical committee. After the nature of the study was explained in detail, informed consent was obtained from all participants. Participants were included if they had not been involved in regular physical activity/diet in the previous 6 months. All subjects were non-smokers and non-pregnancy. Those with type 2 diabetic and other chronic diseases were excluded from the study.

B. Anthropometry

Each subject's body mass and height were measured. Weight was measured to the nearest 100 g using digital scales. Standing height was measured to the nearest 0.1 cm with the use of a wall-mounted stadiometer. Obesity was measured by body mass index (BMI). Percentage of body fat and visceral fat was estimated by bioelectrical impedance method (Omron Body Fat Analyzer, Finland). BMI was calculated as body mass (in kilograms) divided by height squared (in square meters). Waist-to-hip ratio was calculated as abdominal circumference divided by hip circumference as measured to the nearest 0.5 cm with a standard measuring tape. All of these measurements were conducted by the same researcher.

C. Biochemistry and protocol

Blood samples were collected from brachial vein in sitting position at the hormone laboratory after 10-12 hours overnight fast for measure serum resistin. Serum separated immediately after sampling and stored in - 80 centigrade until analysis was performed. Measurement of serum resistin was repeated at 48 hours after lasted session.

All subjects of exercise group participated in an aerobic exercise training intervention of 6 weeks in duration. Exercise sessions started by warm-up then a 30- to 40-min main exercise followed by a cooling-down period. Exercise consisted of treadmill walking or running performed for 30-45 minutes per day, on 3 days per week, at an intensity of 55-70 % of heart rate max. After the last training bout, subjects rested for 48 h, and then fasting serum resistin was measured. Control subjects were instructed to maintain their habitual activities.

D. Data analysis

Statistic analysis was done with SPSS 16.0 for Windows. After calculation of the mean and the standard deviation, normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. Comparisons between the means of each group were done using the independent t-test. Student's paired 't' test was applied to compare the pre and post training values. Statistical significance was accepted at p-value<0.05 or lower.

RESULTS

Baseline (pre-test) and post training resistin levels and anthropometrical indexes of two groups are shown in Table 1. Findings from independent t-test showed that serum resistin and anthropometrical indexes groups were not difference between two groups at baseline (p >0.05, Table 1).

Table 1: Mean and standard deviation of spirometric markers in studied groups.

Variables	Exercise group		Control group	
	Pretest	post-test	Pretest	post-test
Age (year)	37.3 (6.08)	----	38 (2.8)	----
Height (cm)	160.9 (6.30)	----	161 (5.3)	----
Weight (kg)	81 (7.2)	79.4 (7.2)	83.2 (3.73)	83.3 (3.73)
Waist circumference (cm)	108 (7.6)	106 (7.3)	105.5 (4.76)	105.7 (4.98)
Hip circumference (cm)	113 (6.7)	111 (6.3)	107.7 (5.07)	107.6 (5.49)
Abdominal to hip ratio	0.96 (0.08)	0.95 (0.08)	0.98 (0.01)	0.98 (0.02)
BMI (kg/m ²)	31.24 (2.01)	30.64 (1.99)	32.20 (1.3)	32.21 (1.35)
Body fat (%)	45.2 (2.6)	44.5 (2.6)	45.6 (1.85)	45.5 (1.89)
Resistin (ng/ml)	2.83 (1.25)	2.88 (1.71)	2.78 (1.20)	2.70 (1.04)

To confirm of some previous studied that supports relationship between obesity and serum resistin, we also observed that serum resistin is positively related with abdominal obesity in studied subjects ($p = 0.039$, $r = 0.42$, Table 2, Fig 1). Compared to pre-training program, all anthropometrical markers decreased significantly by exercise training in exercise group ($p < 0.05$) but this variables was not changed in control subjects ($p > 0.05$) Serum resistin levels did not change after training when compared with pre-test in exercise group ($p = 0.931$, Fig 2). Serum resistin and all anthropometrical indexes did not change in control subjects ($p > 0.05$).

DISCUSSION

Although several studies have pointed to the role of the lifestyle pattern and diet in obesity phenomenon, recent studies is focusing on the role of cytokines or inflammatory and anti-inflammatory mediators on the mechanisms, influencing the prevalence of obesity and obesity-related diseases. In this regard, several studies have done to improve the inflammatory factors in healthy or ill obese populations who the role of physical activity is prominent among them.

Table 2: Relationship of serum resistin with abdominal circumference of obese subjects at baseline.

		Resistin (ng/ml)	Abdominal (cm)
Resistin (ng/ml)	Pearson Correlation	1	.423 *
	Sig. (2-tailed)		.039
	N	24	24
Abdominal (cm)	Pearson Correlation	.423 *	1
	Sig. (2-tailed)	.039	
	N	24	24

*. Correlation is significant at the 0.05 level (2-tailed).

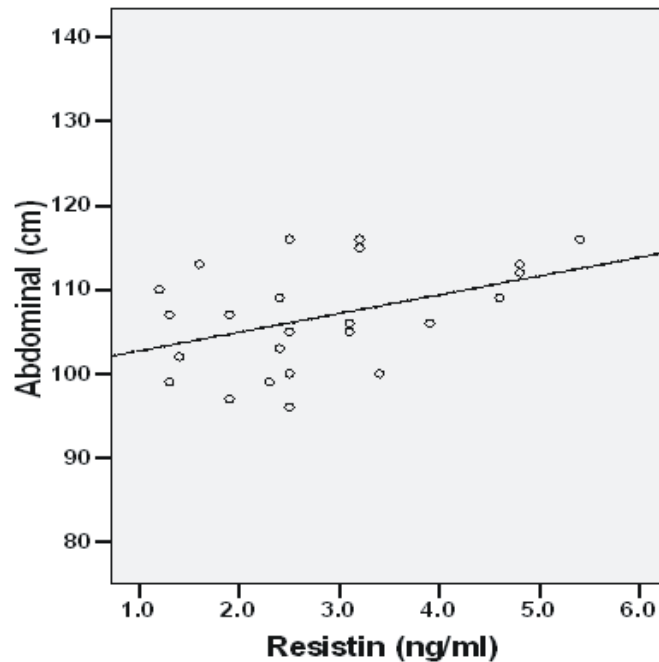


Fig. 1. This Fig shows a significant positive correlation in serum resistin with abdominal circumference of obese subjects at baseline.

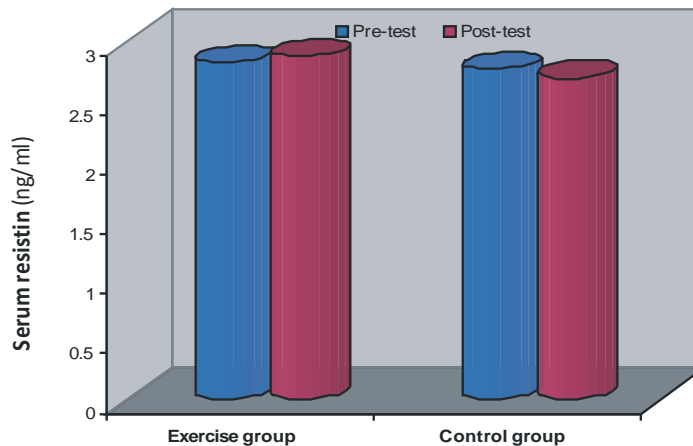


Fig. 2. Serum resistin in pre and post intervention of two groups.

Despite some evidence of anti-inflammatory effects of various training programs, the findings of this study suggest the lack of improvement in the inflammatory mediators in response to aerobic trainings in obese women. In other words, a 6-week aerobic training did not lead to changes in serum levels of resistin in obese women.

Resistin is a peptide mediator, secreted from adipose tissue and other tissues with inflammatory properties. Li, et al showed that obese mouse models have higher resistin levels, compared to their lean counterparts (11). On the other hand, studies have also indicated that higher levels of resistin are independent of obesity or insulin resistance (12, 13, 14). Some of the researchers have reported that its injection in the lean mice leads to increased insulin resistance, while its neutralization leads to improvement of insulin sensitivity in insulin-resisting mice (15). Studies on rodents show higher levels of resistin mRNA expression in abdominal fat stores than in thigh fat stores (16). It has been found that diabetic patients have higher levels of resistin with insulin resistance, compared with normal people (17).

The findings of a study showed a significant positive correlation between resistin and body and anthropometric composition variables such as body mass index, waist to hip ratio, body fat and assessing indices of such as insulin and glucose (18). Hence, it is thought that the weight loss or body fat reduction programs lead to decreased levels of resistin blood circulation. However, in the present study, the weight loss induced by exercise did not lead to any significant change in serum resistin levels in obese women. While 6 weeks of aerobic exercise decreased body weight, body mass index and body fat percentage of the studied obese women, the serum resistin levels were not

significantly changed. No change in serum resistin in the presence of weight loss and body fat after exercise is somewhat controversial, because adipose tissues are the main sources of resistin secretion.

Some studies have reported the lack of change in the resistin or other inflammatory and anti-inflammatory cytokines as a response to long-term training programs, hence supporting the findings of this study. For example, a 6-month change in the life style by combining diet and exercise did not lead to changes in serum resistin levels and some other cytokines such as leptin, adiponectin, Il-6 and TNF- in women with metabolic syndrome (9). In this context, there are also some studies that have reported the lack of correlation between this inflammatory adipocytokine and body mass index in obese women (19). In another study, a non-significant and positive correlation between resistin levels and BMI was observed in healthy individuals (20). In support of these findings, Silha et al have also identified the lack of correlation between resistin and BMI in obese and lean individuals (21).

However, 2 months of aerobic exercise led to the significant decrease in anthropometric indices, improving serum resistin in overweight men with type-2 diabetes (10). In this regard, although some studies have indicated that exercise-induced weight loss is necessary to improve the cytokine or other metabolites (22), some other studies have indicated that a minimum of ten percent weight loss in obese individuals is necessary to improve these variables (23). It should be noted that in this study, although the training program led to a significant reduction in body weight, this reduction was only about 2.5 percent of the individuals' primary weight.

REFERENCES

- Diabetes Prevention Program Research Group (2002). Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med.*, **346**: 393-403.
- Galica, S., Oakhilla, S., Steinberga, G.R. (2010). Adipose tissue as an endocrine organ. *Molecular and Cellular Endocrinology*, **316**: 129-139.
- Shimizu, H., Inoue, K., Mori, M. (2007). The leptin-dependent and -independent melanocortin signaling system: regulation of feeding and energy expenditure. *J Endocrinol*, **193**(1):1-9.
- Uysal KT, Wiesbrock SM, Marino MW, Hotamisligil GS. Protection from obesity-induced insulin resistance in mice lacking TNF- function. *Nature*, 1997; **389**: 610-614.
- Steppan CM, Bailey ST, Bhat S. (2001). The hormone resistin links obesity to diabetes," *Nature*. **409**(6818): 307-312.
- Reilly MP, Lehrke M, Wolfe ML, Rohatgi A, Lazar MA, Rader DJ. (2005). Resistin is an inflammatory marker of atherosclerosis in humans. *Circulation*. **111**(7): 932-9.
- 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.
- Fujinami A, Obayashi H, Ohta K, Ichimura T, Nishimura M. (2004). Enzyme-linked immunosorbent assay for circulating human resistin: resistin concentration in normal subjects and patients with type 2 diabetes. *Clin Chim Acta.*, **339**: 57-6343.
- Oh EG, Bang SY, Kim SH, Hyun SS, Chu SH, Jeon JY, Im JA, Lee JE, Lee MK. (2013). Therapeutic lifestyle modification program reduces plasma levels of the chemokines CRP and MCP-1 in subjects with metabolic syndrome. *Biol Res Nurs*. **15**(1): 48-55.
- 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.
- Lee JH, Bullen JW, Stoyneva VL, Mantzoros CS. (2005). Circulating resistin in lean, obese and insulin-resistant mouse models: lack of association with insulinemia and glycemia. *Am. J. Physiol. Endocrinol. Metab.* **288**: 625-632.
- Ukkola, O. (2002). Resistin a mediator of obesity associated insulin resistance or an innocent bystander?. *European Journal of Endocrinology*, **147**: 571-574.
- Juan CC, Au LC, Fang VS. (2001). Suppressed gene expression of adipocyte resistin in an insulin-resistant rat model probably by elevated free fatty acids. *Biochem Biophys Res Commun.*, **289**: 1328-1333.
- Qin YW, Zheng X, Qiu IJ, Zou DJ. (2003). Serum resistin level in essential hypertension patients with different glucose tolerance. *Diabet Med.*, **20**: 828-831.
- Bastard JP, Maachi M, Lagathu C, Kim MJ, Caron M, Vidal H, Capeau J, Feve B. (2006). Recent advances in the relationship between obesity, inflammation, and insulin resistance. *Eur Cytokine Netw*. **17**(1):4-12.
- Weyer C. (2001). Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and hyperinsulinemia. *J Clin Endocrinol Metab.*, **86**: 1930-1935.
- 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.
- Liu GL, Fu XH, Jiang LH, Ma XC, Yang JY. (2006). Serum resistin concentration and insulin resistance in obese children. *Zhonghua Er Ke Za Zhi*. **44**(2):114-7.
- Janowska J, Zahorska-Markiewicz B, Olszanecka-Glinianowicz M. (2006). Relationship between serum resistin concentration and proinflammatory cytokines in obese women with impaired and normal glucose tolerance. *Metabolism*. **55**(11): 1495-9.
- Schaffler A, Buchler C, Muller ladner U, Herfarth H, Ehling A, Paul G, (2004). Identification of variables influencing resistin serum levels in patients with type 1 and type 2 diabetes mellitus. *Horm Metab Res*; **36**: 702-7.
- Silha JV, Krsek M, Skrha JV, Sucharda P, Nyomba BL, Murphy LJ. (2003). Plasma resistin, adiponectin and leptin levels in lean and obese subjects: correlations with insulin resistance. *Eur J Endocrinol*. **149**(4):331-5.
- 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*. **58**(8):1096-101.
- Johnson WD, Brashear MM, Gupta AK, Rood JC, Ryan DH. (2011). Incremental weight loss improves cardiometabolic risk in extremely obese adults. *Am J Med.*, **124**: 931-938.