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***Research Article***

# ANTI-INFLAMMATORY ADIPOKINE RESISTIN IS NOT ASSOCIATED WITH ANTHROPOMETRICAL MARKERS IN TYPE II DIABETES PATIENTS

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## ABSTRACT

A growing body of evidence supports of systemic inflammation in obesity and diabetes. In this study, the relation of fasting serum resistin as an inflammatory adipokine with anthropometrical markers in twenty eight sedentary males aged 40 ± 4 year and body mass index 32 ± 2 kg/m2 with type II diabetes were determined. Pearson's correlation coefficients were used to determine the correlations between resistin and anthropometrical markers. No significant correlation was found in serum resistin with all anthropometrical markers in studied patients. Based on these finding, it is concluded that the markers of obesity determinatives can not affect serum resistin in diabetic patients.

***Keywords:*** *Body Weight, Diabetes, Inflammation, Obesity*

## INTRODUCTION

Increased prevalence of adipose tissue and risk factors associated with obesity are closely related to prevalence of cardiovascular diseases and type 2 diabetes (Ford, 2005; Yusuf *et al.*, 2005). In terms of health care, obesity and type 2 diabetes are identified as a global epidemic nowadays. Apart from genetic factors and inheritance, scientific evidence clearly identifies obesity as an important factor in incidence of obesity and type 2 diabetes. Obesity also increases incidence of type 2 diabetes due to increased blood glucose and insulin resistance (Maggio *et al.*, 1997). It was previously theorized that adipose tissue act as the only source of fat reserves, which stores fats as triglycerides or fatty acids. However, this theory was replaced with a new hypothesis. It is newly hypothesized that adipose tissue not only plays a pivotal role in lipid and carbohydrates metabolism but also secretes a large number of hormones such as angiotensin, TNF-α, IL-6, adiponectin, leptin and other inflammatory and anti-inflammatory mediators, which cause inflammatory diseases (Engeli *et al.*, 1999; Scherer *et al.*, 1995; Winkler *et al.*, 2003).

Resistin is considered as a new hormone and inflammatory mediator, which secreted from adipocytes. This component belongs to a family of proteins with cysteine-rich carboxyl end. This is also known as RELM (resistin like molecules) or FIZZ (a factor found in the zone) (Steppan *et al.*, 2001; Kim *et al.*, 2001; Holcomb *et al.*, 2000). This hormone was initially isolated as mRNA. Then, it was found out that expression of the latter is repressed by PPARγ agonists. Studies on diabetes models in rodents revealed that these factors are effective in increasing insulin sensitivity (Steppan *et al.*, 2000). Based on this evidence, it can be stated that resistin affects the association between obesity and insulin resistance. Weight loss decreases levels of serum resistin, which is due to appropriate diet or exercise (Jung *et al.*, 2008). Contrary to these findings, several studies suggested no differences in circulating resisten concentrations in lean, obese or diabetic individuals. These also reported that there is no correlation between this hormone and insulin resistance index. In this context, Zhu (2007) and Rinher (2006) argued that resistin levels are not significantly different in obese and lean children (Zou *et al.*, 2008; Reinehr *et al.*, 2006).

Several scientific resources revealed that Resistin is correlated positively and significantly with BMI, WHR, body fat percentage, and glucose and serum insulin levels (Liu *et al.*, 2006). However, several studies suggested that there is no association between serum resistin concentration with each one of such values as BMI, glucose and insulin in obese women (Janowska *et al.*, 2006). Shafler (2004) also found out that there is a poor and positive correlation between resistin levels and BMI in healthy subjects (Schaffler *et al.*, 2004). Silha studied obese and lean individuals in 2003 and demonstrated that there is no correlation between resistin and BMI (Silha *et al.*, 2003). Several recent studies reported that there is no relationship between blood resistin levels and obesity determinant components (Azuma *et al.*, 2003; Lee *et al.*, 2003). It was found out that the findings on the relationship between resistin and anthropometric index and body composition contradict with each other. Hence, in the present study, resistin levels in relation to other anthropometric parameters in type 2 diabetic are determined.

## MATERIALS AND METHODS

Physically inactive, obese men (n = 28) with type II diabetes were recruited for participate in the study by accessible sampling. Participant was a history of type II diabetes at least 3 years. All participants were inactive or sedentary, non-smoker and non-alcoholic. All participants gave their informed written consent before participation in study. Potential participants were excluded from the study if they reported a history of heart disease, cancer, respiratory and kidney diseases. Those patients unable to avoid taking hypoglycemic drugs or other therapeutic drugs within 12 hours before blood sampling were excluded.

### Anthropometric measurements

Body weight, height, waist circumference and % body fat measurements were obtained by standard methods. 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. Body mass index (BMI) was calculated by dividing body mass (kg) by height in metres squared (m2). Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter. Waist to hip circumference ratio was measured by dividing the abdominal circumference into that of the hip. Body composition monitor (BF508-Omron made in Finland) with a precision error of less than 100 g was used to measure weight and body fat percentage of the subjects.

### Laboratory measurements

All subjects were asked to attend in hematology lab after an overnight fat between 8:00 a.m. and 9:00. Venous blood samples were obtained of each patient for calculating serum resistin by Eliza method. The Intra- assay coefficient of variation and sensitivity of the method were 3.4% and 0.033 ng/mL.

### Data analysis

Data were analyzed by computer using SPSS software version 15.0. We verified normal distribution of variables with a Kolmogorov–Smirnov test. Pearson's correlation coefficients were used to evaluate the correlations between serum resistin and anthropometrical markers in studied subjects. Significance was accepted at $P < 0.05$.

## RESULTS

In this study, relationship between serum resistin with anthropometrical markers in males with type 2 diabetes were determined. Anthropometrical features of the studied patients are showed in table 1. All data represented by mean and standard deviation. In studied subjects, serum resistin and glucose and insulin resistance was 1.91 ± 1.19 ng/ml and 238 ±69 and 4.63 ±1.27respectively.

Based on Pearson correlation method, serum resistin was not correlated with all anthropometrical markers such as body weight ($p = 0.27$, $r = 0.22$), BMI ($p = 0.46$, $r = 0.15$), abdominal obesity ($p = 0.15$, $r = 0.28$) and body fat percentage ($p = 0.39$, $r = 0.17$) (Fig 1).

**Table 1: Anthropometric characteristics of the study participants**

| | N | Minimum | Maximum | Mean | Std. Deviation |
|---|---|---|---|---|---|
| Age (Year) | 28 | 32 | 47 | 40.71 | 4.026 |
| Height (cm) | 28 | 168 | 181 | 173.71 | 3.321 |
| Weight (kg) | 28 | 88 | 106 | 97.00 | 5.193 |
| Abdominal (cm) | 28 | 94 | 118 | 106.25 | 7.085 |
| Hip (cm) | 28 | 98 | 114 | 104.79 | 4.677 |
| WHO | 28 | .90 | 1.11 | 1.0139 | .04263 |
| BMI (kg/m2) | 28 | 30 | 36 | 32.15 | 1.671 |
| Body fat (%) | 28 | 28 | 35 | 31.46 | 1.520 |

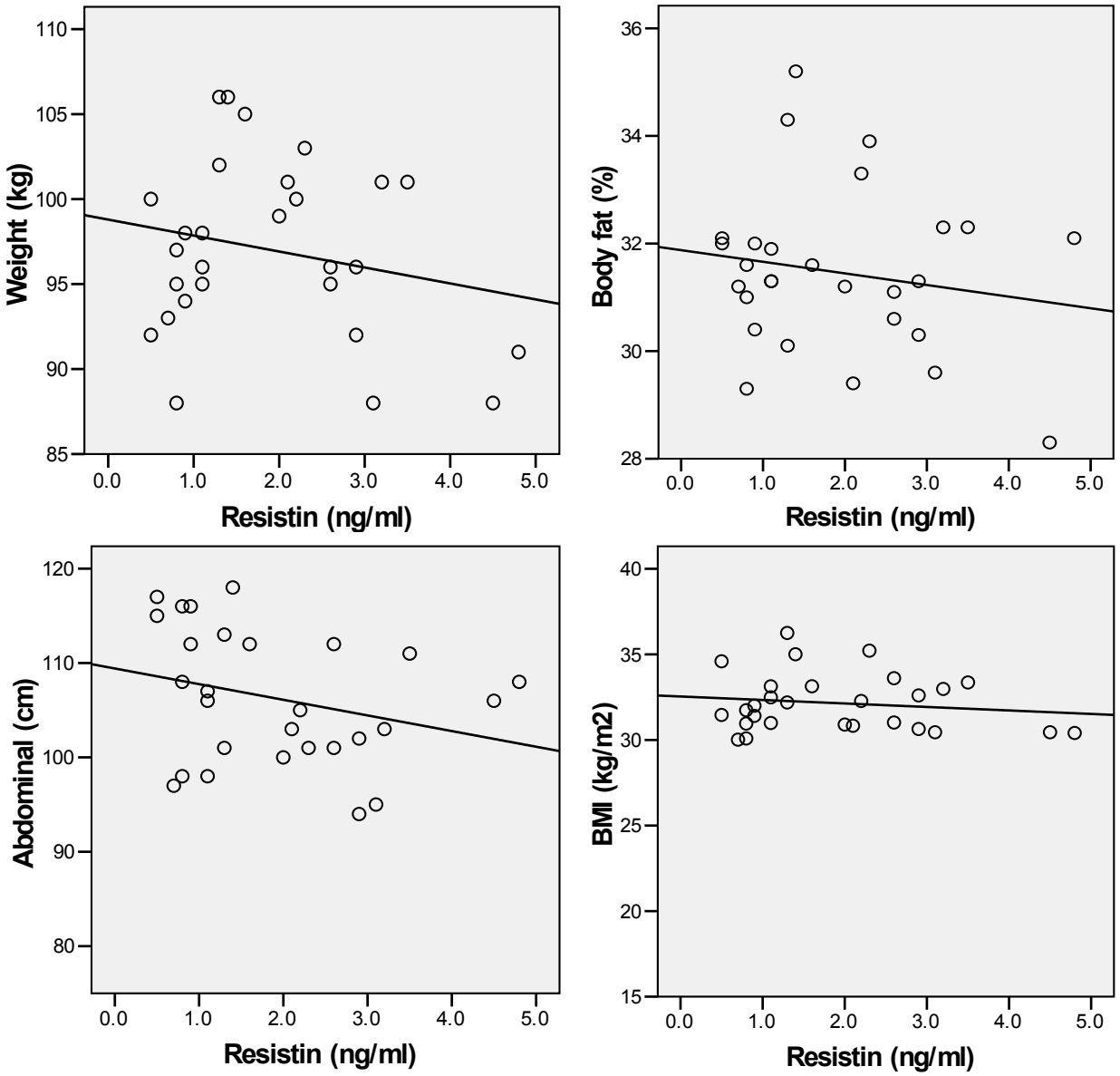


**Figure 1: The serum resistin in relation to anthropometrical markers in studied patients. No significant correlation was found between resistin with body weight, body fat (%), BMI and abdominal circumference in studied diabetes patients**

## DISCUSSION

The direct correlation of serum resistin levels with obesity and insulin resistance was observed in many previous studies (Ursula *et al.,* 2004' Rajala *et al.,* 2003; Banerjiee *et al.,* 2004). Since type II diabetes is also associated primarily with obesity and body fat levels, it can be stated that there is a close relationship between serum resistin levels and this disease. Hence, a direct correlation between serum resistin levels and anthropometric indices or body composition is predictable in these patients. However, despite this evidence, the findings obtained from the present study suggested that there is no correlation between serum resistin and anthropometric indices in these patients.

It is controversial whether resistin serum levels in type 2 diabetes are independent of body weight and fat levels or not. However, this is not the first study that reported that there is no correlation between anthropometric indices or body composition and resistin levels in obese or diabetic individuals. Rather, our findings contradict with studies on obese individuals who underwent weight loss due to diet. Nevertheless, several studies reported that resistin levels dropped or did not change in obese patients although they lost weight (Azuma *et al.,* 2003; Moschen *et al.,* 2009; de Luis *et al.,* 2011). Other studies reported that there is no association between serum resistin and obesity or insulin resistance parameters (Jain *et al.,* 2009; Youn *et al.,* 2004; Reilly *et al.,* 2005). Nevertheless, the association between resistin levels and visceral, intra-aortic and cardiac adipose tissues were reported in several cross-sectional studies (Liu *et al.,* 2006).

Although resistin affects the relationship between obesity and insulin resistance in rodents, the role of resistin in humans is not known precisely. Several studies examined pathophysiologic significance of changes in circulating resistin over the last few years. Although early studies on rodents (Steppan *et al.,* 2004) and humans (Silha *et al.,* 2003) addressed the potential relationship between circulating resistin levels and insulin resistance, several recent studies on humans suggested that there is no association between resistin and obesity and insulin resistance (Azuma *et al.,* 2003; Lee *et al.,* 2003). On the other hand, Liu (2006) showed that resistin is correlated positively and significantly with BMI, WHR, body fat percentage, glucose and serum insulin levels (Liu *et al.,* 2006). However, Janoska (2006) pointed out that serum resistin levels in obese women are not correlated with any values such as BMI, glucose, and insulin (Janowska *et al.,* 2006). In addition, Shafler (2004) demonstrated a poor and positive correlation between resistin levels and BMI in healthy subjects while no association was observed between resistin levels and BMI in diabetic individuals (Schaffler *et al.,* 2004). Silha demonstrated that there is no relationship between resistin and BMI in obese and lean individuals in 2003 (Silha *et al.,* 2003). However, Velfing showed that glucose, insulin and GH are involved in resistin secretion (Wölfing *et al.,* 2008).

Resistin either is less expressed or is not expressed in human adipocytes due to reduced genomic binding sites in nuclear receptors (Tomaru *et al.,* 2009). This showed that diet has a significant impact on resistin regulation in rodents. Resistin expression is reduced in adipose tissue during starvation; moreover, re-feeding in mice leads to increased expression of resistin (Azuma *et al.,* 2003; Rajala *et al.,* 2002; Rajala *et al.,* 2004). In the present study, serum resistin was measured in diabetic individuals in fasting state. In addition, lack of correlation of serum resistin with anthropometric indices may be attributed to the measurement time since several researchers pointed out that serum resistin reduced in fasting mice. In other words, fasting serum resistin levels decreased in mice (Park *et al.,* 2013). However, lack of relationship between anthropometric indices or body composition and resistin may be attributed to the low number of samples studied. This is considered as one limitations of this study.

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