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 the role 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 Reinehr (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 \((m^2)\). 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 ±1.27 respectively.

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

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Minimum</th>
<th>Maximum</th>
<th>Mean</th>
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<tr>
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<td>28</td>
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<td>Body fat (%)</td>
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<td>28</td>
<td>35</td>
<td>31.46</td>
<td>1.520</td>
</tr>
</tbody>
</table>

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.
**REFERENCES**


Indian Journal of Fundamental and Applied Life Sciences ISSN: 2231-6345 (Online)
An Open Access, Online International Journal Available at http://www.cibtech.org/ijfls.htm

Research Article


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