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Research Article

TYPE II DIABETES DOES NOT AFFECT SERUM RESISTIN IN PRESENCE OBESITY

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ABSTRACT

Growing bodies of evidence in obesity have implicated adipocytokines in the development of insulin resistance and type 2 diabetes. The aim of the present work was to compare serum resistin between adult obese males with or without type II diabetes matched for age and body weight. For this purpose, serum resistin and glucose concentration were measured in fourteen males with type 2 diabetes and same number males healthy men. All subjects were obese and non-trained. Comparisons in each variable between two groups were done using the independent t-test. Serum resistin was not significantly difference between two groups ($p = 0.43$). We found that fasting glucose was significantly higher in diabetes patients than healthy subjects ($p = 0.000$). Serum resistin was not correlated with body weight in two groups ($p \geq 0.000$). Based on this data, we can say that diabetes can not affect serum resistin in obese subjects.

Keywords: *Type II Diabetes, Obesity, Resistin*

INTRODUCTION

The adipocytokines secreted by the adipose tissue play a major role in the metabolism, immune system and inflammation. Resistin is an adipocytokine with a molecular weight of 12.5 kDa. In addition to adipocytes, this adipocytokine is secreted in muscles, pancreatic islet, mononuclear cells and human placenta (Reilly *et al.*, 2005). In rodents, resistin is primarily secreted by adipocytes. It has been identified as a bridge between obesity and insulin resistance (Steppan *et al.*, 2001). However, resistin is primarily secreted by macrophages in humans (Patel *et al.*, 2003). It noteworthy that resistin is associated with inflammation. On the other hand, obesity, Type 2 diabetes and cardiovascular disease have recently been recognized as chronic inflammatory disorders. Such inflammatory diseases may be associated with pro-inflammatory cytokines and adipocytokines such as resistin (Hotamisligil, 2006).

According to Lee *et al*, obese mouse models showed higher resistin levels compared with the lean counterparts (Lee *et al.*, 2005). Given the close relationship between resistin and insulin resistance in mice, several clinical studies examined the possible relationship between resistin and insulin resistance in diabetic and non-diabetic obese individuals. Obesity and Type 2 diabetes are associated with chronic inflammation in white adipose tissue. On the other hand, resistin is mainly secreted in macrophages. Accordingly, hyper-resistin may play a major role in the pathophysiology of these disorders. Although resistin affects the relationship between obesity and insulin resistance in rodents, its role in humans is not known precisely.

In recent years, several studies have examined the pathophysiology role of circulating resistin. Although early studies on rodents (Steppan *et al.*, 2001) and humans (Silha *et al.*, 2003) indicate the potential relationship between circulating resistin levels and insulin resistance, recent studies on humans suggest

the lack of relationship between resistin, obesity and insulin resistance (Azuma *et al.,* 2003; Lee *et al.,* 2003). Nevertheless, there are conflicting studies on the relationship between resistin and insulin resistance and Type 2 diabetes (Lee *et al.,* 2003; Reinehr *et al.,* 2006; Liu *et al.,* 2006; Minn *et al.,* 2003; Brown *et al.,* 2007; Jamurtas *et al.,* 2006). Some studies demonstrate the direct relationship between resistin and insulin resistance in obese or diabetic patients, but others show no relationship between resistin and insulin resistance. Based on this inconsistency, the present study aimed to compare serum resistin levels between adult obese males with or without type II diabetes.

MATERIALS AND METHODS

This study was aimed to compare serum resistin between adult obese males with or without type II diabetes. Subject were fourteen adults men with type II diabetes (age, 40 ± 5 kg; BMI, 31.6 ± 2.3 kg/m2) and fourteen healthy males (age, 38 ± 4 kg; BMI, 31 ± 1.60) that participated in study by accidentally. All subjects were non-athletes and no smoker. Those that participated in involved in regular physical activity in the previous 6 months were excluded. Potential participants were excluded from the study if they reported smoking or had a history of acute or chronic respiratory infections, neuromuscular disease, and cardiopulmonary disease. After the nature of the study was explained in detail, informed consent was obtained from all participants.

Anthropometric measurements of height, weight, percent body fat, and circumference measurements were taken of all subjects of two groups. Body weight and height were measured with a standard physician's scale and a stadiometer, respectively when subjects were in a fasting state. BMI was calculated as weight (in kilograms) divided by the square of height (in meters). Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter. Visceral fat and body fat percentage was determined using body composition monitor (OMRON, Finland). Systolic and diastolic blood pressure was measured using the left arm after the subject had been sitting comfortably for 5 min, using an oscillometric device (Alpikado, Japan).

Fasting blood samples were taken after an overnight fast to determine serum resistin and glucose concentration. Glucose was determined by the oxidase method (Pars Azmoun, Tehran, Iran). Serum resistin was determined by ELISA method (Biovendor-Laboratoria medicina a.s. Czech). Intra- assay and inter-assay coefficient of variation of the method were 2.8% and 5.1 respectively for resistin.

Statistical Analysis

Data were analyzed by computer using the Statistical Package for Social Sciences (SPSS) for Windows, version 11.5. Comparisons in means of each variable between two groups were done using the independent t-test. The association between serum resistin concentration and body weight were assessed using Pearson's correlation coefficient. A p-value of less than 0.05 was considered to be statistically significant.

RESULTS

In this study, serum resistin was compared between males with type II diabetes and those with non-diabetes. As above mentioned, all subjects of diabetes and non-diabetes were obese. Table 1 show the descriptive anthropometric and biochemical features of the study groups. Based on independent T test, all anthropometrical markers were same between two groups (p – 0.000). No significant differences was found between two groups in serum resistin (p= 0.43, table 2). Fasting glucose was significantly higher in diabetes patients than non-diabetes individuals (p = 0.000, table 2). Serum resistin was not correlated with body weight in two groups (p = 0.11 diabetes, p = 0.68 non-diabetes).

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

	Diabetes=1, Non-Diabetes=2	N	Mean	Std. Deviation	Std. Error Mean
Age (year)	1	14	40.43	5.019	1.341
	2	14	38.43	4.345	1.161
Height (cm)	1	14	173.79	3.906	1.044
	2	14	175.29	3.688	.986
Weight (kg)	1	14	95.50	6.285	1.680
	2	14	95.36	5.624	1.503
Systole	1	14	13.29	1.978	.529
	2	14	12.86	.949	.254
Diastole	1	14	8.57	.852	.228
	2	14	9.00	.961	.257
Abdominal (cm)	1	14	106.29	7.248	1.937
	2	14	106.86	5.289	1.414
Hip (cm)	1	14	104.71	4.140	1.107
	2	14	105.93	3.751	1.003
WHO	1	14	1.0150	.04381	.01171
	2	14	1.0086	.02825	.00755
BMI (kg/m2)	1	14	31.64	2.265	.605
	2	14	31.03	1.595	.426
Body Fat (%)	1	14	30.87	2.507	.670
	2	14	31.74	2.731	.730
Visceral Fat	1	14	12.86	1.956	.523
	2	14	13.36	1.499	.401
Resistin (ng/ml)	1	14	1.821	1.1396	.3046
	2	14	2.257	1.6965	.4534
Glucose (mg/dL)	1	14	221.50	62.850	16.797
	2	14	101.36	10.043	2.684

Table 2: Independent samples test of resistin and glucose in two groups

		t-test for Equality of Means						
		t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
							Lower	Upper
Resistin (ng/ml)	Equal variances assume	-.798	26	.432	-.4357	.5462	-1.5585	.6870
	Equal variances not assumed	-.798	22.748	.433	-.4357	.5462	-1.5663	.6949
Glucose (mg/dL)	Equal variances assume	7.063	26	.000	120.143	17.010	85.177	155.108
	Equal variances not assumed	7.063	13.663	.000	120.143	17.010	83.575	156.711

Serum resistin was not differences between two groups, while glucose concentration in diabetes patients was higher than non-diabetes subjects

DISCUSSION

Discrepancies in baseline levels of peptide mediators such as inflammatory or anti-inflammatory cytokines between healthy subjects and patients, obese and lean or athletic and non-athletic individuals have been previously reported (Oda, 2013). Some studies reported similar inflammatory cytokines for obese individuals and those with normal body weight (Owecki *et al.*, 2010). However, some studies found significant differences in the levels of peptide mediators such as adiponectin and TNF-α in diabetics and non-diabetics (Hivert *et al.*, 2008). Hence, similar fasting serum resistin levels in diabetic and non-diabetic subjects in the present study is not a new result. Indeed, the findings of the present study showed no significant difference in serum resistin levels between adult men with Type 2 diabetes and healthy men. In this regard, some previous studies support the direct relationship between resistin levels, obesity and insulin resistance. In other words, resistin plays a major role in relationship between obesity and insulin resistance in diabetics through pro-inflammatory pathways (Mojiminiyi *et al.*, 2007). The findings of another study showed the impact of plasma resistin changes in the development of Type 2 diabetes in obese individuals with an emphasis on the higher levels of serum resistin in type 2 diabetics as compared with healthy controls (Al-Harithy *et al.*, 2005). Some other studies also support the role of resistin in visceral obesity, obesity-induced insulin resistance and incidence of type 2 diabetes (Chanchay *et al.*, 2006; Codoñer-Franch *et al.*, 2014).

Despite these statements, the findings of another study showed that the serum resistin levels are not associated with insulin sensitivity, lipid profile and body mass index in Type 2 diabetics. In other words, serum resistin levels in type 2 diabetics were similar with healthy subjects (Stejskal *et al.*, 2003). According to the results of another study, it seems that circulating resistin level does not play an important role in insulin resistance or metabolic syndrome in humans (Utzschneider *et al.*, 2005). According to Hasegawa *et al* (2005), although serum resistin levels are increased in diabetics, this increase is not related to body fat or insulin resistance determinant symptoms (Hasegawa *et al.*, 2005).

The basic mechanisms influencing the chronic phenomena like diabetes or heart-cardiovascular disease and inflammatory or anti-inflammatory cytokine levels are not yet known. In addition, the potential impact of impairment in the levels of cytokines in the incidence or severity of these diseases is not also yet known. However, since most of these diseases are rooted in obesity, it appears that obesity is among most important factors involved in impaired mediators in type 2 diabetics or those with cardiovascular disease. It should be noted that both diabetic and non-diabetic obese subjects in this study are men. In other words, the analogical factor in both groups is obesity. Hence, according to the findings of the present study and some previous studies, it appears that obesity affects the serum resistin levels in obese subjects not diabetes as compared with those with normal body weight. According to the results of the present study on obese individuals, serum resistin levels are not affected by the presence of diabetes.

REFERENCES

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

Azuma K, Katsukawa F, Oguchi S, Murata M, Yamazaki H, Shimada A and Saruta T (2003). Correlation between serum resistin level and adiposity in obese individuals. *Obesity Research* **11**(8) 997-1001.

Brown JE, Onyango DJ and Dunmore SJ (2007). Resistin down-regulates insulin receptor expression, and modulates cell viability in rodent pancreatic beta-cells. *FEBS Letters* **581**(17) 3273-6.

Chanchay S, Tungtrongchitr R, Harnroongroj T, Phonrat B, Rungseesakorn O, Paksanont S, Pooudong S, Saowakontha S and Varongchayakul C (2006). Plasma resistin, insulin concentration in non-diabetic and diabetic, overweight/obese Thai. *International Journal for Vitamin and Nutrition Research* **76**(3) 125-31.

Codoñer-Franch P, Tavárez-Alonso S, Porcar-Almela M, Navarro-Solera M, Arilla-Codoñer Á and Alonso-Iglesias E (2014). Plasma resistin levels are associated with homocysteine, endothelial activation, and nitrosative stress in obese youths. *Clinical Biochemistry* **47**(1-2) 44-8.

Hasegawa G, Ohta M, Ichida Y, Obayashi H, Shigeta M, Yamasaki M, Fukui M, Yoshikawa T and Nakamura N (2005). Increased serum resistin levels in patients with type 2 diabetes are not linked with markers of insulin resistance and adiposity. *Acta Diabetologica* **42**(2) 104-9.

Hivert MF, Sullivan LM, Fox CS, Nathan DM, D'Agostino RB Sr, Wilson PW and Meigs JB (2008). Associations of adiponectin, resistin, and tumor necrosis factor-alpha with insulin resistance. *Journal of Clinical Endocrinology and Metabolism* **93**(8) 3165-72.

Hotamisligil GS (2006). Inflammation and metabolic disorders. *Nature* **444** 860-7.

Jamurtas AZ, Theocharis V, Koukoulis G, Stakias N, Fatouros IG, Kouretas D and Koutedakis Y (2006). The effects of acute exercise on serum adiponectin and resistin levels and their relation to insulin sensitivity in overweight males. *European Journal of Applied Physiology* **97**(1) 122-6.

Lee JH, Bullen JW, Stoyneva VL and Mantzoros CS (2005). Circulating resistin in lean, obese and insulin-resistant mouse models: lack of association with insulinemia and glycemia. *American Journal of Physiology - Endocrinology and Metabolism* **288** 625–632

Lee JH, Chan JL, Yiannakouris N, Kontogianni M, Estrada E and Seip R (2003). Circulating resistin levels are not associated with obesity or insulin resistance in humans and are not regulated by fasting or leptin administration: cross-sectional and interventional studies in normal, insulin resistant, and diabetic subjects. *Journal of Clinical Endocrinology and Metabolism* **88** 4848-4856.

Liu GL, Fu XH, Jiang LH, Ma XC and Yang JY (2006). Serum resistin concentration and insulin resistance in obese children]. *Zhonghua Er Ke Za Zhi* **44**(2) 114-7.

Minn AH, Patterson NB, Pack S, Hoffmann SC, Gavrilova O, Vinson C, Harlan DM and Shalev A (2003). Resistin is expressed in pancreatic islets. *Biochemical and Biophysical Research Communications* **310**(2) 641–645.

Mojiminiyi OA and Abdella NA (2007). Associations of resistin with inflammation and insulin resistance in patients with type 2 diabetes mellitus. *Scandinavian Journal of Clinical and Laboratory Investigation* **67**(2) 215-25.

Oda E (2013). High-sensitivity C-reactive protein and white blood cell count equally predict development of the metabolic syndrome in a Japanese health screening population. *Acta Diabetologica* **50**(4) 633-8.

Owecki M1, Nikisch E, Miczke A, Pupek-Musialik D and Sowiński J (2010). Serum resistin is related to plasma HDL cholesterol and inversely correlated with LDL cholesterol in diabetic and obese humans. *Neuroendocrinology Letters* **31(5)** 673-8.

Patel L, Buckels AC, Kinghorn IJ, Murdock PR, Holbrook JD, Plumpton C, Macphee CH and Smith SA (2003). Resistin is expressed in human macrophages and directly regulated by PPAR gamma activators. *Biochemical and Biophysical Research Communications* **300** 472-6.

Reilly MP, Lehrke M, Wolfe ML, Rohatgi A, Lazar MA and Rader DJ (2005). Resistin is an inflammatory marker of atherosclerosis in humans. *Circulation* **111**(7) 932-9.

Reinehr T, Roth CL, Menke T and Andler W (2006). Resistin concentrations before and after weight loss in obese children. *International Journal of Obesity (Lond)* **30**(2) 297-301.

Silha JV, Krsek M, Skrha JV, Sucharda P, Nyomba BL and Murphy LJ (2003). Plasma resistin, adiponectin and leptin levels in lean and obese subjects: correlations with insulin resistance. *European Journal of Endocrinology* **149**(4) 331-5.

Stejskal D, Adamovská S, Bartek J, Juráková R and 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 Republic* **147**(1) 63-9.

Steppan CM, Balley ST, Bhat S, Brown EJ, Banerjee RR and Wright CM (2001). The hormone resistin links obesity to diabetes. *Nature* **409** 307-312.

Utzschneider KM, Carr DB, Tong J, Wallace TM, Hull RL, Zraika S, Xiao Q, Mistry JS, Retzlaff BM, Knopp RH and Kahn SE (2005). Resistin is not associated with insulin sensitivity or the metabolic syndrome in humans. *Diabetologia* **48**(11) 2330-3.