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*Full Length Research Paper*

# The relationship between plasma omentin-1 levels and insulin resistance in newly diagnosed type 2 diabetıc women

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**In addition to its role in energy storage, adipose tissue produces several hormones and cytokines termed adipokines that have widespread effects on carbohydrate and lipid metabolism. Omentin-1 is a newly identified adipokine that is highly and selectively expressed in visceral adipose tissue relative to subcutaneous adipose tissue. In some recent studies, it was shown to be decreased in obese and in insulin resistant female patients. In this study, intending to increase their knowledge about omentin-1 and its relation with type 2 diabetes mellitus, insulin resistance and obesity, they planned to point out the relationship between serum omentin-1 levels and insulin resistance in newly diagnosed type 2 diabetic patients. The study included 80 newly diagnosed female type 2 diabetic patients and 40 age matched female control subjects. Diabetic group had significantly lower plasma omentin-1 levels than the control group ($p < 0.01$). Both the diabetic and control groups who were insulin resistant had significantly lower omentin-1 levels ($p < 0.009$ and $p < 0.05$ respectively) than the groups who were not insulin resistant. Positive weak correlations were obtained between age, high density lipoprotein cholesterol and omentin-1 levels, and negative weak correlations between body mass index, fasting blood glucose, post prandial blood glucose, HbA1c, fasting insulin, model assessment insulin resistance index and omentin-1 levels. In conclusion, omentin-1 levels were significantly low in diabetic patients and as insulin resistance worsened omentin-1 decreased. The data may point toward a role of omentin-1 in insulin resistance and type 2 diabetes mellitus.**

**Key words:** Omentin-1, diabetes mellitus, insulin resistance.

## INTRODUCTION

The precise relationship between adiposity and insulin resistance is complex and undefined (Grundy, 2004; Despres et al., 2008; Hofso et al., 2009; Bakhai, 2008; Shah et al., 2008). Epidemiological studies often report an association between obesity and mortality due to increased rates of cardiovascular and cerebrovascular diseases and diabetes (Kannel, 1985; Larsson, 1991; Chung et al., 2009) Adipose tissue has been shown to secrete a variety of bioactive peptides, called adipokines that can potentially impact on glucose and lipid metabolism. These adipokines include adiponectin, leptin, resistin, visfatin, interleukin-6, and tumor necrosis factor- (Hotamı lıgil et al., 1993, Depres et al., 2008, Shah et al., 2008). Recently, a new protein omentin (also named omentin-1, intelectin, intelectin-1, endothelial lectin and intestinal lactoferrin receptor) has been identified as a major visceral (omental) fat secretory adipokine.

Visceral obesity is reportedly more pathogenic than subcutaneous obesity in promoting insulin resistance, type 2 diabetes, and cardiovascular disease. In addition, visceral fat accumulation is associated with accumulation of triglycerides in muscle and liver, contributing to insulin resistance in these tissues. Omentin-1 was shown to be

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**Abbreviations: FPG,** fasting plasma glucose; **FI,** insulin; **HbA1c,** hemoglobin A1c; **HDL-C,** high density lipoprotein cholesterol; **LDL -C,** low density lipoprotein cholesterol; **TG,** triglyceride; **BMI,** body mass index; **PPPG,** postprandial plasma glucose; **SBP,** systolic blood pressure; **DBP,** systolic blood pressure; **WHR,** waist- hip ratio; **HOMA-IR,** homeostasis model assessment insulin resistance index.

predominantly expressed in visceral adipose tissue, and was among the first molecules known to exhibit such a dramatic difference in *gene* expression between the two major fat depots. As a secretory factor, omentin-1 may be a novel hormone that is likely to act as both an endocrine factor to modulate systemic metabolism, including insulin action in subcutaneous adipocytes, and an autocrine and paracrine factor to regulate visceral adipose biology locally (Yang et al., 2006, Gualillo et al., 2007).

Studies have demonstrated that omentin-1 enhances insulin action by stimulating insulin-mediated glucose uptake by subcutaneous as well as omental adipocytes *in vitro* (Yang et al., 2006). There are few sudies about obesity, diabetes mellitus and omentin-1. Lean subjects had significantly higher plasma omentin-1 levels than obese and overweight subjects (Souza–Batista et al., 2007; Tan et al., 2008a). Decreased plasma omentin-1 levels were reported in type 1 (Tan et al., 2008b) and type 2 diabetes (Cai et al., 2009; Pan et al., 2010) and in patients with impaired glucose regulation ( Cai et al., 2009; Pan et al., 2010) Keeping in mind that omentin-1 levels may be predictive of the metabolic consequences or co-morbidities associated with obesity and glucose metabolism, they planned to analyse the relationship of plasma omentin-1 with some parameters of adiposity, insulin resistance and plasma lipid profile in newly diagnosed type 2 diabetic patients.

## PATIENTS AND METHODS

### Patients

A total of 80 female newly diagnosed type 2 diabetics aged from 45 - 65 years, were recruited from the outpatient Clinic of Ankara Education and Research Hospital from February 2009 to June 2009. Type 2 DM was diagnosed according to the criteria of WHO (WHO, Geneva, 1999). 40 aged matched female subjects formed the control group. As omentin-1 levels are reportedly higher in women than in men (Souza-Batista et al., 2007) in order to obtain a homogenous group we included only female subjects in our study. Patients with male gender, conditions which may effect metabolic parameters (such as polycystic ovary syndrome or thyroid dysfunctions in history or present), pregnancy, chronic diseases, infection, coronary artery disease were excluded. None of the female subjects were on any medications for at least 6 months before the study including oral contraceptives, glucocorticoids, ovulation induction agents, anti-diabetic and anti-obesity drugs, estrogenic, anti-androgenic, anti-hypertensive or anti-hyperlipidemic medication.

After detailed physical examination, body weight and height were measured in all subjects. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters. Body fat was estimated by Tanita body composition analyser TBF -300 after the subjects rested for 30 min. Blood pressure was measured with a sphygmomanometer, after a 5 min rest in the semi-sitting position. Blood pressure was determined at least three times at the right upper arm, and the mean was used in the analysis. Patients who were taking anti-hypertensive drugs or patients with mean blood pressure levels 140/90 mmHg were assumed to be hypertensive and excluded. Blood was withdrawn after 12 h of overnight fasting, at 08.30 a.m. for fasting plasma glucose (FPG), insulin (FI), hemoglobin A1c (HbA1c) serum total and high density lipoprotein cholesterol (HDL-C), triglyceride (TG), and omentin-1 levels. Another blood sample was taken for postprandial plasma glucose (PPPG) 2 h after breakfast. The local ethics commitee approved this study and all the subjects gave written informed consent.

### Laboratory methods

Plasma glucose, total cholesterol, triglyceride (TG) and HDL cholesterol concentrations were determined by enzymocalorimetric spectrophotometric method in a Roche/Hitachi molecular PP autoanalyser. Low density lipoprotein cholesterol (LDL-C) was calculated by the Friedewald Formula (LDL-C: Total cholesterol – HDL-TG/5). Insulin was measured by means of DRG Diagnostics (DRG Instruments GmbH, Germany) ELISA kits. HbA1c level was measured by TOSOH G7 HPLC system.

The estimate of insulin resistance by homeostasis model assessment insulin resistance index (HOMA-IR) was calculated as fasting plasma insulin ( unite / ml) x fasting plasma glucose (mmol /l ) / 22. 5. As in normal person HOMA level was stated to be $< 2.7$, it was chosen as a cut-point for insulin resistance (Wallace et al., 2007). As omentin has two isolated forms, omentin- 1 is also called as omentin, omentin-2 is secreted from Paneth cells into the intestinal lumen, so it is slightly detected in the serum (Souza et al., 2007). Therefore, they examined serum levels of omentin-1. For the measurements of omentin-1, after fasting, blood samples were drawn, were centrifuged at 4000 cycle / min in 30 min. Plasma was then stored at -75˚C, in two different tubes. Plasma omentin-1 levels were assayed by a commercial USCNLIFE (Chinese) ELISA kit.

### Statistical analysis

Calculations were performed using SPSS version 10,1. Data are presented as mean ± SD. Student t- test was used to compare the groups in a parametric way (For homogeneously distributed data). Non parametric Mann Whitney U test was used for non homogen distributed data. For determining the correlations within the parameters Pearson correlation analysis was made. They used two- tailed test, a p value of $< 0.05$ was considered as statistically significant.

## RESULTS

This study was performed with 80 newly diagnosed type 2 diabetic females (Group 1), and 40 female control subjects (Group 2). All the demographic and laboratory findings of the groups were compared and shown in Table 1. SBP, DBP, BMI, FBG, PPBG, HbA1c of the diabetic group were found statistically higher than the control group (respectively, $p < 0.01$, $p < 0.03$, $p < 0.01$, $p < 0.01$, $p < 0.01$ $p < 0.01$) . In the diabetic patients total cholesterol, TG, LDL-C, FI, HOMA-IR, levels were also statistically higher than the control group (respectively, $p < 0.01$, $p < 0.01$, $p < 0.09$, $p < 0.005$, and $p < 0.001$). They could not find any difference between the age, body fat, HDL-C levels of the two groups. The omentin-1 levels of the diabetic patients were statistically lower than the control subjects (Table 1) ($p < 0.001$). They then, grouped the diabetic patient and control subject groups according to their HOMA-IR levels as HOMA-IR<2.7 and

**Table 1.** Characteristics of group 1 vs group 2.

| | GROUP 1 (n = 80) | GROUP 2 (n = 40) | P |
|---|---|---|---|
| Age (yr) | 52.8± 10.7 | 54.7 ± 8.2 | NS |
| SBP (mmHg) | 136.2 ± 19.5 | 22.6± 17.2 | <0.01 |
| DBP (mmHg) | 86.2± 17.9 | 79.0 ± 6.9 | <0.03 |
| BMI ( kg/m$^2$) | 30.5 ± 4.8 | 28.1 ± 5.7 | <0.01 |
| Body fat (%) | 33.1 ± 9.6 | 30.3 ± 8.9 | NS |
| FBG (mg/dl) | 199.5 ± 80.7 | 88.0± 16.2 | <0.01 |
| PPBG (mg/dl) | 297.9± 100.1 | 111.2 ± 17.1 | <0.01 |
| HbA1c (%) | 8.7± 2.6 | 5.6± 0.3 | <0.01 |
| Cholesterol(mg/dl) | 226.6 ± 70.1 | 185.5 ± 37.2 | <0.01 |
| TG(mg/dl) | 243.8 ± 58.3 | 140.2 ± 69.2 | <0.01 |
| LDL-C(mg/dl) | 132.8 ± 47.2 | 110.8 ± 32.7 | <0.09 |
| HDL-C (mg/dl) | 45.7± 11.4 | 46.0 ± 9.2 | NS |
| FI ( u/ml) | 16.0 ± 1.0 | 10.4 ± 2.2 | <0.005 |
| HOMA-IR | 7.8± 1.0 | 2.2± 0.8 | <0.001 |
| Omentin-1(ng/ml) | 307.9± 153.1 | 461.0± 153.2 | <0.001 |

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index, WHR: waist- hip ratio, FBG: fasting blood glucose, PPBG: post prandial blood glucose, TG: triglyceride, LDL: low density lipoprotein cholesterol, HDL: high density lipoprotein cholesterol, FI: fasting insulin, HOMA-IR: Homeostasis model assessment insulin resistance index, Data are presented as mean ± SD. NS: nonsignificant. Group 1: diabetic group, Group 2: control group.

**Table 2.** Characteristics of the diabetic patients whose HOMA-IR was < 2.7and 2.7.

| | HOMA-IR < 2.7 (n = 11) | HOMA-IR 2.7 (n = 69) | P |
|---|---|---|---|
| Age (yr) | 53.2± 11.6 | 52.7 ± 10.6 | NS |
| SBP (mmHg) | 126.8± 14.3 | 137.7± 19.8 | NS |
| DBP (mmHg) | 87.5 ±34.0 | 86.0 ± 14.2 | NS |
| BMI ( kg/m$^2$) | 27.3± 3.3 | 31.0± 4.8 | <0.01 |
| Body fat (%) | 29.9± 7.2 | 33.6 ± 9.9 | NS |
| FBG (mg/dl) | 170.9± 82.4 | 204.1± 80.1 | NS |
| PPBG (mg/dl) | 272.9 ± 105.0 | 301.9± 99.5 | NS |
| HbA1c (%) | 8.7± 2.6 | 8.7 ± 2.5 | NS |
| Cholesterol(mg/dl) | 201.3± 59.4 | 230.6± 71.2 | NS |
| TG (mg/dl) | 175.0± 89.6 | 254.8± 27.7 | <0.05 |
| LDL-C(mg/dl) | 124.5± 53.2 | 134.1± 46.4 | <0.05 |
| HDL-C (mg/dl) | 42.0± 11.9 | 46.3 ± 11.2 | NS |
| FI ( u/ml) | 6.5± 3.3 | 17.5± 1.1 | <0.02 |
| Omentin-1 (ng/ml) | 419.9 ± 174.9 | 290.1±142.9 | <0.009 |

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index, WHR: waist- hip ratio, FBG: fasting blood glucose, PPBG: post prandial blood glucose, TG: triglyceride, LDL: low density lipoprotein cholesterol, HDL: high density lipoprotein cholesterol, FI: fasting insulin, HOMA-IR: Homeostasis model assessment insulin resistance index. Data are presented as mean ± SD. NS: nonsignificant.

HOMA-IR 2.7. The data of diabetic and control females were shown in Tables 2 and 3.

BMI was found to be elevated in diabetic patients with HOMA- IR 2.7(p<0,01). Age, SBP, DBP, body fat, FBG, PPBG, HbA1c, cholesterol, TG, LDL-C, HDL-C levels of the HOMA-IR < 2.7 and HOMA-IR 2.7 diabetic patients were statistically insignificant (Table 2). In the group with HOMA-IR 2.7 the diabetic patients had statistically lower omentin-1 levels than the diabetic patients with HOMA-IR < 2.7 (p < 0.009) (Table 2). Age, SBP, DBP,BMI, body fat, FBG, PPBG, HbA1c, cholesterol, TG, LDL-C, HDL-C, FI levels of the HOMA-IR < 2.7 vs HOMA-IR 2.7

**Table 3.** Characteristics of the control group whose HOMA-IR was < 2.7and 2.7.

| | HOMA-IR < 2.7 (n = 31) | HOMA-IR 2.7 (n = 9) | P |
|---|---|---|---|
| Age (yr) | 54.9± 9.1 | 54.2 ± 4.0 | NS |
| SBP (mmHg) | 122.0± 15.2 | 124.4 ± 24.0 | NS |
| DBP (mmHg) | 79.8 ± 18.9 | 78.2 ± 24.0 | NS |
| BMI ($kg/m^2$) | 28.0± 5.9 | 28.7 ± 5.1 | NS |
| Body fat (%) | 30.1± 9.0 | 30.8 ± 8.9 | NS |
| FBG (mg/dl) | 86.3 ± 17.3 | 93.8± 10.1 | NS |
| PPBG (mg/dl) | 110.3 ± 17.9 | 114.3 ± 14.3 | NS |
| HbA1c (%) | 5.6 ± 0.3 | 5.6± 0.2 | NS |
| Cholesterol(mg/dl) | 182.4± 34.0 | 196.1± 47.4 | NS |
| TG (mg/dl) | 134.3± 67.8 | 160.5 ± 74.5 | NS |
| LDL-C(mg/dl) | 108.6± 32.6 | 118.2 ± 34.1 | **NS** |
| HDL-C (mg/dl) | 46.1± 9.9 | 45.6 ± 7.0 | NS |
| FI ( u/ml) | 7.0 ± 2.7 | 7.1± 1.2 | NS |
| Omentin-1 (ng/ml) | 479.1± 97.1 | 398.7± 137.0 | <0.05 |

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index, WHR: waist- hip ratio, FBG: fasting blood glucose, PPBG: post prandial blood glucose, TG: triglyceride, LDL: low density lipoprotein cholesterol, HDL: high density lipoprotein cholesterol, FI: fasting insulin, HOMA-IR: Homeostasis model assessment insulin resistance index. Data are presented as mean ± SD. NS: nonsignificant.

**Table 4.** A summary of correlation analysis among all females (n: 120).

| | | Age | SBP | DBP | BMI | Body fat | FBG | PPBG | Cholesterol | TG | LDL-C | HDL-C | HbA1c | FI | HOMA-IR |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| omentin-1r | | 0.260 | -0.036 | -0.002 | -0.217 | 0.017 | -0.443 | -0.379 | -0.115 | -0.145 | -0.096 | 0.265 | -0.39 | -0.378 | -0.485 |
| | P | 0.004 | 0.696 | 0.987 | 0.017 | 0.857 | 0.000 | 0.000 | 0.211 | 0.114 | 0.298 | 0.003 | 0.000 | 0.000 | 0.000 |

SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index, WHR: waist- hip ratio, FBG: fasting blood glucose, PPBG: post prandial blood glucose, TG: triglyceride, LDL: low density lipoprotein cholesterol, HDL: high density lipoprotein cholesterol, FI: fasting insulin, HOMA-IR: Homeostasis model assessment insulin resistance index.

control subjects were statistically insignificant (Table 3). In the group with HOMA-IR 2.7 the control subjects had statistically lower omentin-1 levels than the control subjects with HOMA-IR < 2.7 ($p < 0.05$) (Table 3).

Then, they mixed all patient and control females and correlation analysis was made among he paremeters. Positive weak correlations were obtained between age (r:0.26 $p < 0.004$), HDL-C (r:0.26 $p < 0.003$) and omentin-1 levels, and negative weak correlations between BMI (r:-0.21 $p < 0.017$), FBG (r:-0.44 $p < 0.001$) PPBG (r:-0.37 $p < 0.001$), HbA1c (r:-0.39 $p < 0.001$), FI (r:-0.37 $p < 0.001$), HOMA-IR (r:-0.48 $p < 0.001$) and omentin-1 levels were obtained (Table 4). Significant correlations were demonstrated in bold within the table.

## DISCUSSION

Over the last two decades studies have re-emphasized the notion put forward in 1947 by Vague that obesity is not a homogeneous condition and that the regional distribution of adipose tissue is important to understand the relation of obesity with disturbances of lipid and glucose

metabolism (Vague, 1947). Many studies have shown that excess fat in the upper part of the body considered by Vague as 'android obesity' correlates with increased mortality and risk for various metabolic disorders more often than the 'gynoid' type of fat distrubition (Bouchard et al., 1993; Wajchenberg, 2000). Although, the cause-effect association has not yet been definitely established the available evidence indicates that visceral fat is an important link between many facets of these metabolic disturbances. In 2003, a new protein named omentin-1 was described and reported to be expressed specifically in human omental adipose tissue. It was then found that omentin-1 is predominantly expressed in visceral but not in subcutaneous adipose tissue, with the omentin-1 mRNA being 150 times higher in the visceral adipose tissue (Yang et al., 2006).

Lean subjects had significantly higher plasma omentin-1 levels than obese and overweight subjects (Souza–Batista et al., 2007; Tan et al., 2008a). In addition, higher plasma omentin-1 levels were detected in women compared with men. Omentin-1 *gene* expression was also decreased with obesity. It is interesting to note that the omentin-1 *gene* is localized on a chromosomal region of 1q22–q23, where it was reported a presence of linkage to type 2 diabetes in various populations. These data may suggest that omentin-1 may be a positional candidate *gene* for type 2 diabetes susceptibility in humans. How omentin-1 levels are influenced by glucose levels and visa versa glucose levels by omentin-1 levels warrant elucidation (Tan et al., 2008a; Yang et al., 2006; Wurm et al., 2007). Decreased omentin-1 levels were reported in subjects with impaired glucose regulation (Pan et al., 2010), type 1 (Tan et al., 2008 b) and type 2 diabetic individuals (Cai et al., 2009; Pan et al., 2010).

In this study, female diabetic patients had statistically significant lower omentin-1 levels than control females. The diabetic patients and control females with high HOMA- IR had lower omentin- 1 levels. Between the four groups of diabetic and control groups with HOMA-IR low and high, the lowest omentin-1 levels were found in HOMA-IR high diabetic patients. The results demonstrated that omentin -1 levels decreased in diabetic individuals and decreased further when diabetes mellitus was combined with insulin resistance. They also found positive correlation between omentin-1 and age, HDL-C levels of all the females. For the present time, it is diffucult to estimate the importance of omentin- 1 in getting older, but as HDL- C is concerned omentin-1 may be a protective marker for cardiovascular disease as was shown in the literature (Souza–Batista et al., 2007, Cai et al., 2009). Like a few studies in the literature about omentin-1, plasma omentin-1 levels of our subjects were inversely correlated with BMI (Tan et al., 2008b, Cai et al., 2009, Souza–Batista et al., 2007; Pan et al., 2010). In this study, in concordance with the previous studies, fasting and post prandial glucose, HbA1c, HOMA-IR and fasting insulin levels were also found to be negatively correlated with omentin-1 levels (Tan et al., 2008a; Tan et al., 2008b; Souza–Batista et al., 2007; Cai et al., 2009; Pan et al., 2010) . For the time being, it is difficult to say whether high glucose and insulin levels are the cause or the result of low omentin-1 levels and with which mechanisms they effect omentin-1 levels. Further studies are needed.

In conclusion, the study showed that omentin-1 levels are low in type 2 diabetics and insulin resistant females. Since type 2 diabetes mellitus is closely related to visceral adipose tissue amount and diabetes has declared to be a state of inflammation it was reasonable to further investigate the role of omentin-1 in type 2 diabetic patients. According to the study, it may be said that glucose and insulin levels as well as insulin resistance may have a repressive effect on omentin-1 levels. Decreased omentin-1 levels may contribute to the underlying pathophysiology of insulin resistance and diabetes mellitus. Furthermor, as they can declare that an important marker of serum omentin-1 level is insulin resistance, as was shown as HOMA-IR in their study, decreased omentin- 1 levels may contribute to the underlying pathology of insulin resistance. Future studies probably with bigger sample size will be required to address the link of omentin-1 with metabolic disturbances such as obesity, insulin resistance and the regulation of omentin-1 in diabetic patients.

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