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Serum concentration of visfatin in obese women

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Abstract

The aim of the present study was to determine serum concentrations of visfatin in obese women in comparison to normal-weight controls. Study subjects were 21 obese women without additional disease (age, 29.0 ± 4.9 years; body mass index, 37.1 ± 6.1 kg/m^2) and 16 healthy, normal-weight women (age, 29.9 ± 5.4 years; body mass index, 22.5 ± 1.7 kg/m^2). Body composition was measured by bioimpedance. Serum concentrations of visfatin were assayed with an enzyme-linked immunosorbent assay kit (Phoenix Pharmaceuticals, Burlingame, CA). Insulin was determined by radioimmunoassay and glucose by colorimetric method. Serum concentration of visfatin was significantly higher in obese women when compared to controls. Positive correlations between serum concentrations of visfatin and insulin in the obese group were found. In the control group, we observed positive correlations between serum concentrations of visfatin and glucose. In conclusion, the observed increase of visfatin in obesity may be a counterregulation preventing further glucose increase.

1. Introduction

Adipose tissue releases a lot of adipokines such as tumor necrosis factor α (TNF-α), interleukin 6 (IL-6), leptin, adiponectin, and resistin [1]. These cytokines participate in induction of insulin resistance [2]. Production, release, and serum concentrations of TNF-α, IL-6, leptin, and resistin significantly increase in obesity [3,4].

Recently, a new adipokine, visfatin, was described. Visfatin corresponds to a protein identified previously as pre–B-cell colony-enhancing factor, a 52-kd cytokine expressed in lymphocytes. Visfatin is predominantly secreted from visceral adipose tissue. However, Varma et al [5] revealed that visfatin is highly expressed in subcutaneous adipose tissue in lean, more insulin-sensitive subjects. Therefore, they concluded that expression of visfatin in visceral adipose tissue and subcutaneous adipose tissue is regulated oppositely with body mass index (BMI). Experimental studies showed that this cytokine participates in glucose homeostasis because it has glucose-lowering effect [6].

So far, only a few studies assessed visfatin gene expression in visceral adipose tissue and plasma visfatin concentrations in humans, and their results are divergent. For example, Berndt et al [7] and Hammarstedt et al [8] reported that serum concentration of visfatin is increased in obesity. Haider et al [9] also described elevated plasma visfatin concentrations in morbidly obese subjects and its reduction after weight loss. On the other hand, Pagano et al [10] revealed that plasma visfatin and its messenger RNA in subcutaneous adipose tissue were significantly lower in obese subjects. However, significantly higher visfatin messenger RNA was found in visceral adipose tissue.

The aim of present study was to determine serum concentration of visfatin in obese and normal-weight women.

2. Materials and methods

The study group consisted of 21 obese women, aged 29.0 ± 4.9 years. The control group consisted of 16 lean volunteers, aged 29.9 ± 5.4 years. The characteristics of the study and control groups are presented in Table 1.

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Table 1
Subjects' characteristics

	Obese	Control	*P*
Weight (kg)	99.5 ± 15.0	60.9 ± 6.8	.0001
BMI (kg/m^2)	37.1 ± 6.1	22.5 ± 1.7	.0001
Body fat (kg)	52.7 ± 15.0	16.4 ± 4.9	.0001
Body fat (%)	52.4 ± 9.2	26.6 ± 6.3	.0001
Fat-free mass (kg)	46.8 ± 8.2	44.5 ± 5.1	NS
Fat-free mass (%)	47.6 ± 9.5	73.4 ± 7.5	.0001
WHR	0.79 ± 0.05	0.84 ± 0.03	NS

WHR indicates waist-to-hip ratio.

All obese subjects included in the study were diagnosed as having simple obesity without additional diseases.

The study was conducted after obtaining informed consent from all the subjects. The study was approved by the local ethical committee.

Body weight and height were measured and BMI was calculated. The reference interval of BMI is defined as 19 to 24.9 kg/m^2 (controls) and obesity as a BMI of more than 30 kg/m^2 (study group). Body composition was determined by bioimpedance analysis with the use of the Bodystat analyzer (Bodystat 1500, Bodystat Ltd, Douglas, British Isles).

Six- to eight-milliliter samples of venous blood were collected in the morning, after an overnight fast. After clot formation, the samples were centrifuged (1000*g*) at room temperature for 10 minutes. The obtained serum was drawn into a few plastic vials and stored at −80°C until the time of assay.

Plasma glucose, cholesterol, high-density lipoprotein (HDL) cholesterol, and triglicerydes were determined by enzymatic procedure using the commercially available test kit (Cormay, Warsaw, Poland). Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald formula.

Insulin was determined by radioimmunoassay (DPC Diagnostic Products, Los Angeles, CA) with a lower limit of sensitivity of 1.2 μIU/mL and intra- and interassay coefficients of variations of 5.2% and 5.8%, respectively. Insulin resistance was assessed on the basis of fasting serum concentrations of glucose and insulin; the homeostasis model assessment (HOMA) index was calculated using the formula: HOMA = fasting serum concentration of insulin (μIU/mL) × fasting serum concentration of glucose (mmol/L)/22.5. The reference range of HOMA is less than 2.77.

Serum concentrations of visfatin were assayed using a commercial EIA kit (Phoenix Pharmaceuticals) with a lower limit of sensitivity of 2.63 ng/mL and intra-and interassay coefficients of variations of 5.2% and 5.8%, respectively.

Data were analyzed using *t* test and Pearson correlation analysis. Values are presented as mean ± SD. *P* values of less than .05 were considered to be statistically significant.

3. Results and discussion

Serum concentrations of visfatin and insulin were significantly higher in obese women when compared to controls ($P < .05$ and $P < .00001$, respectively) (Table 2).

We did not show correlation between serum concentration of visfatin and BMI, waist-to-hip ratio, and percentage of body fat in both obese and lean women.

Our results are in accordance with the report of Berndt et al [7], which showed that serum concentration of visfatin is increased in obesity. However, in contrast to the results obtained by Berndt et al [7], we did not show correlation between serum concentration of visfatin and BMI and percentage of body fat in both obese and lean women.

Previous experimental study demonstrated [5] that visfatin participates in glucose homeostasis. Therefore, we assessed serum concentrations of glucose and insulin in both groups, and insulin resistance was assessed on the basis of HOMA index. All women in the study had HOMA index values in the reference range.

In obese women we observed significant positive correlation between serum concentrations of visfatin and glucose ($r = 0.52$, $P = .02$) (Fig. 1), and in lean women, significant positive correlation was found between serum concentrations of visfatin and insulin ($r = 0.51$, $P = .045$) (Fig. 2).

In contrast, Berndt et al [7] found no significant correlation between visfatin plasma concentrations and fasting plasma insulin and glucose concentrations. On the other hand, results obtained by Hammarstedt et al [8] revealed circulating visfatin levels were about 2-fold higher in the type 2 diabetic subjects than in the nondiabetic subjects. It, to some extent, may be the effect of differences between values of BMI in their study groups (type 2 diabetic subjects were obese and nondiabetic subjects were overweight). Chen et al [11] also observed increased serum concentration of visfatin in patients with type 2 diabetes mellitus. Our results may suggest that visfatin compensates for the impairment of insulin action in the early stage of development of insulin resistance. On the other hand, experimental studies showed that TNF-α and IL-6 downregulated visfatin expression in 3T3-L1 adipocytes [11-13]. Further studies are necessary to clarify whether and what interaction appears between the action of visfatin and insulin resistance–inducing adipocytokines in human.

There were no differences in serum concentrations of total cholesterol, LDL cholesterol, triglicerydes, and glucose

Table 2
Serum concentrations of visfatin, insulin, glucose, and lipids

	Obese	Control	*P*
Visfatin (ng/mL)	78.6 ± 44.0	50.6 ± 26.0	.03
Insulin (μIU/mL)	12.0 ± 4.7	4.8 ± 3.1	.00001
Glucose (mg/dL)	94.5 ± 9.2	90.0 ± 8.2	NS
HOMA	2.74 ± 0.2	1.1 ± 0.7	.01
Total cholesterol (mg/dL)	178.2 ± 28.5	174.6 ± 32.0	NS
LDL cholesterol (mg/dL)	115.4 ± 29.7	102.3 ± 28.2	NS
HDL cholesterol (mg/dL)	50.3 ± 8.5	61.2 ± 11.6	.002
Triglycerides (mg/dL)	115.5 ± 104.3	84.3 ± 31.4	NS

NS indicates not significant.

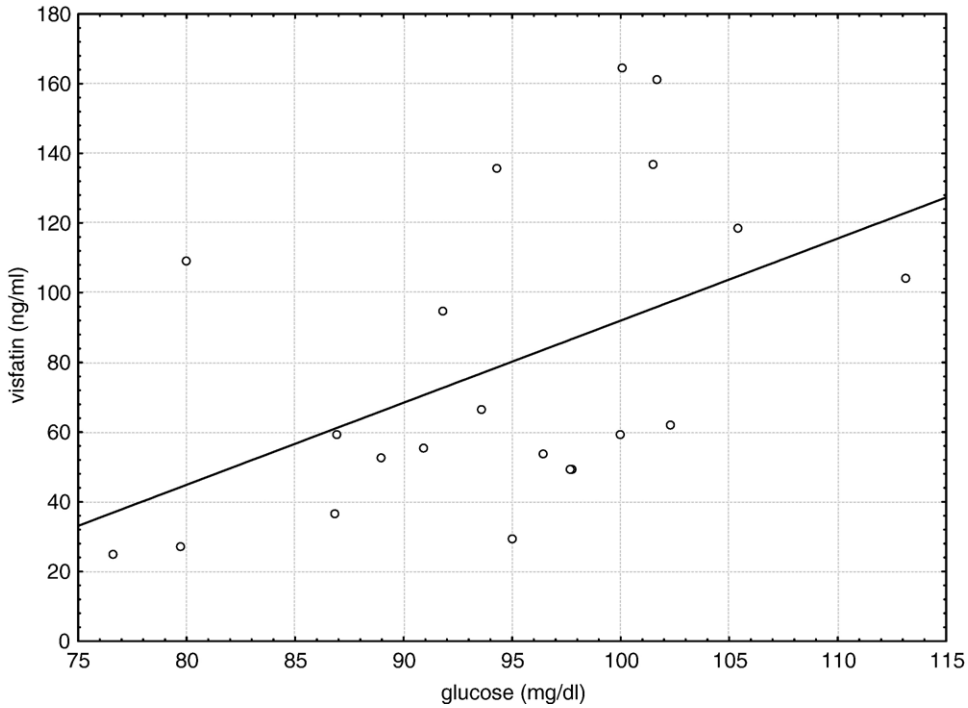

Fig. 1. Correlations of plasma visfatin and glucose in obese group.

between study and control groups. Serum concentration of HDL cholesterol was significantly lower in obese women when compared to controls (Table 2).

We did not observe a correlation between serum concentrations of visfatin and lipids in both the obese and lean groups.

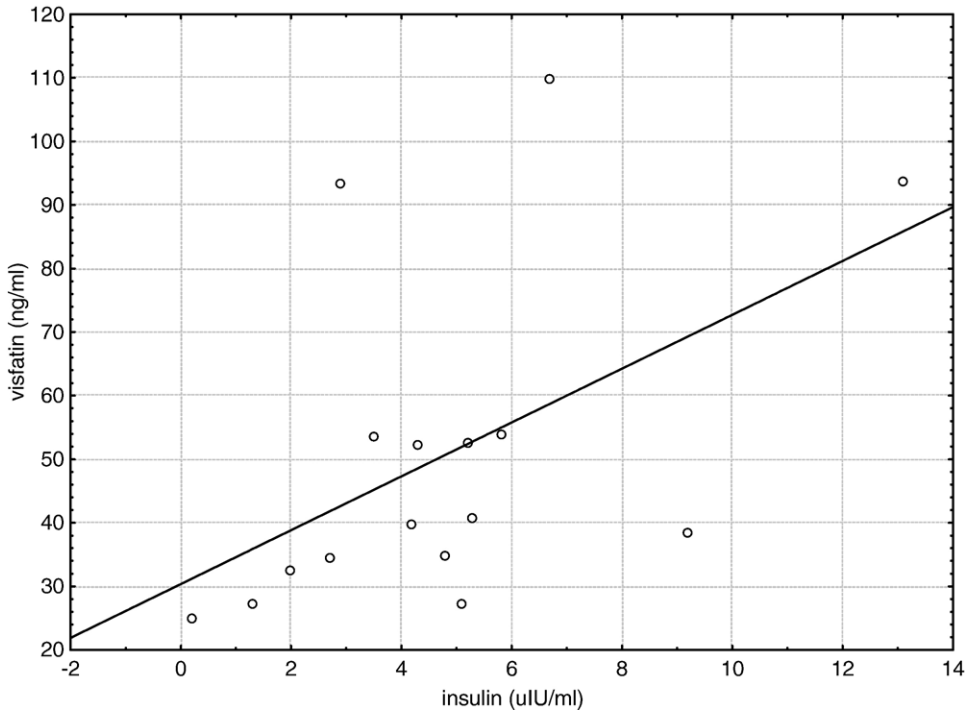

Fig. 2. Correlations of plasma visfatin and insulin in control group.

We did not observe a correlation between serum concentrations of visfatin and lipids in both obese and lean groups.

4. Conclusions

The observed increase of visfatin in obesity may be a counterregulation preventing further glucose increase.

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