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Plasma visfatin and tumor necrosis factor-alpha (TNF-α) levels in metabolic syndrome

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Abstract

Background: Experimental studies have shown that tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) downregulate visfatin gene expression in adipocytes. On the other hand, the induction of cytokine production by visfatin in leucocytes and monocytes has also been described.

Aim: To assess the possible interrelation between plasma concentrations of visfatin and TNF-α and TNF soluble receptor in obese women fulfilling, or not, the criteria of metabolic syndrome (MS).

Methods: Ninety two obese women were included in the study. Metabolic syndrome, based on IDF criteria (2005) was diagnosed in 71 subjects (mean age 53 ± 9 years; body mass index 39.1 ± 5.6 kg/m^2, waist circumference 109.6 ± 11.4 cm). The remaining 21 formed the non-MS subgroup (mean age 52 ± 9 years, body mass index 36.3 ± 5.2 kg/m^2, waist circumference 104.7 ± 11.0 cm). Fourteen healthy normal weight women served as controls. In all subjects, body composition was assessed by the bioimpedance method.

Results: In the MS subgroup, but not in the non-MS subgroup, visfatin levels were significantly higher than in controls. We did not observe any significant difference in plasma concentrations of visfatin, TNF-α or sTNFRs between the MS subgroup and the non-MS subgroup. Only in the MS subgroup and in the combined analysis of all study subgroups did plasma visfatin concentrations correlate significantly with TNF-α levels ($R = 0.31$, $p = 0.01$, $R = 0.21$, $p = 0.03$; respectively). Additionally, in the MS subgroup there was a positive correlation between visfatin levels and insulin resistance ($R = 0.53$, $p = 0.01$).

Conclusions: Our findings suggest that visfatin in metabolic syndrome should be regarded as a proinflammatory factor indirectly favouring the development of insulin resistance.

Key words: visfatin, TNF-α, sTNFRs, obesity, metabolic syndrome

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INTRODUCTION

Adipose tissue is an endocrine organ producing and secreting biologically active factors — adipokines, among them tumor necrosis factor-alpha (TNF-α) [1], interleukin-6 (IL-6) [2], leptin [3], adiponectin [4], resistin [5], visfatin [6], vaspin [7] and omentin [8]. Some of these adipokines, like TNF-α, IL-6, leptin and resistin, are overexpressed in adipose tissue of obese subjects, and have well-known proinflammatory and insulin resistance-increasing properties [9–11].

Visfatin, a recently described adipokine, predominantly expressed in visceral adipose tissue, is upregulated in obese animals and humans [6, 12]. Fukuhara et al. [6] revealed that

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recombinant visfatin has insulin-mimetic activity. It has been shown *in vitro* and *in vivo* that visfatin binds to the insulin receptor type 3 and activates tyrosine phosphorylation as well as phosphorylation of insulin receptor substrate-1 and -2, increasing glucose uptake. Increased plasma visfatin levels have also been described in subjects with type 2 diabetes mellitus [13]. Thus elevated visfatin production in obesity seems to be a compensatory, although unfortunately insufficient, response in obesity-induced insulin resistance [14].

It has been shown that glucocorticoids stimulate visfatin synthesis in cultured 3T3-L1 adipocytes, while TNF-α, IL-6, growth hormone, and β-adrenergic receptor agonists inhibit its synthesis [15, 16]. On the other hand, visfatin underregulates, in dose-dependent manner, the production of TNF-α and IL-6 in human monocytes [17, 18], suggesting its proinflammatory activity. These findings prompt the question: is visfatin a definitely 'beneficial' adipokine? May its proinflammatory activity be counterbalanced by insulin sensitising properties in obese subjects?

The aim of this study was to assess the possible link between plasma concentrations of visfatin and TNF system in obese women with and without metabolic syndrome (MS).

METHODS

Study group

Ninety two obese women with stable weight over the previous three months were included in the study. Their obesity lasted for several years. Patients with a weight loss or gain of more than 2 kg during the previous six months were not enrolled. Exclusion criteria were: type 2 diabetes, treated arterial hypertension, gastrointestinal disease, any medication, pregnancy, acute and chronic inflammatory diseases, drug abuse, smoking and consumption of more than two alcoholic drinks per week. The study was approved by the local ethics committee. All subjects gave their informed consent for participation in the study.

Seventy one subjects fulfilled the criteria of MS (IDF 2005), while 21 others did not (non-MS group). Fourteen lean and healthy women without obesity served as controls. The characteristics of the study subgroups are presented in Table 1.

In all subjects, anthropometric measurements (body weight, height, waist circumference) were performed, blood pressure was measured, and body mass index (BMI) was calculated according to the standard formula. Body composition was assessed by the bioimpedance method using a Bodystat analyser (United Kingdom).

Laboratory procedures

The 6–8 mL samples of venous blood were collected in the morning between 8am and 9am, after overnight fasting (15 h), according to the recommendations of the kit manufacturers. Serum and plasma samples were stored at –80°C.

Plasma glucose, total cholesterol, LDL and HDL cholesterol as well as triglycerides were estimated by colorimetric methods using a commercially available test kit (Roche, Switzerland).

Serum insulin concentration was determined by radioimmunoassay (DPC Diagnostic Products Corporation, Los Angeles, CA, USA) with a lower limit of sensitivity of 1.2 μIU/mL and intra- and inter-assay coefficients of variations of 5.2% and 5.8%, respectively. Based on fasting serum concentrations of glucose and insulin, insulin resistance HOMA-IR index was calculated using the standard formula: HOMA-IR = = fasting concentration of insulin (μIU/mL) × fasting concentration of glucose (mmol/L)/22.5.

The plasma concentrations of visfatin were assayed by a commercial ELISA kit from Phoenix Pharmaceuticals (Burlingame, CA, USA) with a lower limit of sensitivity of 2.63 ng/mL and intra- and inter-assay coefficients of variations of 5.2% and 5.8%, respectively.

Table 1. Characteristics of obese women, fulfilling (MS) or not (non-MS) the criteria of metabolic syndrome, and controls

	MS	Non-MS	Controls
N	71	21	14
Age [years]	53.0 ± 9.0#	52.0 ± 9.0#	38.0 ± 8.0
Body mass [kg]	100.9 ± 15.0#	94.3 ± 13.9#	61.9 ± 7.2
Body mass index [kg/m^2]	39.1 ± 5.6#	36.3 ± 5.2#	23.1 ± 2.7
Body fat [kg]	52.0 ± 12.8*#	45.2 ± 10.7#	20.6 ± 5.4
Body fat [%]	50.8 ± 6.3#	47.4 ± 6.9#	32.7 ± 5.6
Fat-free mass [kg]	48.9 ± 5.8#	49.1 ± 9.0#	41.4 ± 3.2
Fat-free mass [%]	49.2 ± 6.5#	52.6 ± 6.7#	67.3 ± 5.6
Waist circumference [cm]	110.0 ± 11.0#	105.0 ± 11.0#	78.0 ± 10.0
Systolic blood pressure [mm Hg]	145 ± 21#	132 ± 14#	120 ± 15
Diastolic blood pressure [mm Hg]	87 ± 12	80 ± 10	80 ± 10

*$p < 0.05$ *vs* non-MS; #$p < 0.001$ *vs* controls

Table 2. Plasma lipids, glucose and insulin levels in obese women, fulfilling (MS) or not (non-MS) the criteria of metabolic syndrome, and in controls

	MS	Non-MS	Controls
Total cholesterol [mg/dL]	237.4 ± 50.9*	205.3 ± 44.3	222.5 ± 38.0
LDL-cholesterol [mg/dL]	160.3 ± 58.6	127.1 ± 43.7	142.4 ± 41.0
HDL-cholesterol [mg/dL]	46.4 ± 13.4**$^{\#\#}$	59.9 ± 15.3	61.7 ± 12.8
Triglycerides [mg/dL]	154.3 ± 71.2***$^{\#\#}$	85.8 ± 25.3	90.3 ± 47.7
Glucose [mg/dL]	114.3 ± 26.9***$^{\#}$	89.2 ± 9.1	93.1 ± 15.8
Insulin [μIU/mL]	16.6 ± 10.9**$^{\#}$	8.8 ± 4.6	6.7 ± 3.3
HOMA-IR	5.5 ± 4.3***$^{\#\#}$	2.0 ± 1.2	1.6 ± 0.9

*p < 0.05, **p < 0.01, ***p < 0.005 *vs* non-MS; $^{\#}$p < 0.01, $^{\#\#}$p < 0.001 *vs* controls

Table 3. Plasma concentrations of adipokines in obese women, fulfilling (MS) or not (non-MS) the criteria of metabolic syndrome, and in controls

	MS	Non-MS	Controls
Visfatin [ng/mL	33.6 ± 12.6$^{\#}$	29.8 ± 8.8	27.9 ± 10.1
TNF-α [pg/mL]	6.3 ± 1.9	6.4 ± 5.5	5.6 ± 2.0
sTNFR1 [pg/mL]	1,372 ± 533	1,250 ± 505$^{\#}$	1,502 ± 468
sTNFR2 [pg/mL]	2,104 ± 682	1,871 ± 540	2,123 ± 438

$^{\#}$p < 0.05 *vs* controls

The plasma concentrations of TNF-α and sTNFRs were measured using a commercially available highly sensitive ELISA kit from R&D Systems (Minneapolis, MN, USA). The sensitivity of the TNF-α assay was less than 0.18 pg/mL. Mean intra-assay coefficient of variance was < 14.4% and mean inter-assay coefficient of variance was < 18.7%. The sensitivity of the sTNFR1 and sTNFR2 assays was typically less than 0.77 pg/mL and 0.6 pg/mL, respectively. Mean intra-assay coefficients of variances were < 3.6% and 2.6% respectively, and mean inter-assay coefficients of variances were < 3.7% and 3.5%, respectively.

Statistical analysis

All statistical analyses were performed with the use of Statistica 8.0 software. Results are presented as means ± SD. The Mann-Whitney *U* test was used for subgroup comparisons. The Spearman rank-order test was used to calculate the univariate correlation coefficients between the analysed parameters. The results were considered as significant with a p value of less than 0.05.

RESULTS

Both groups of obese women, i.e. MS and non-MS, were of similar age, and had comparable body mass, BMI, waist circumference, and fat free mass (Table 1). Only the content of fat mass expressed in kilograms was greater in the subgroup with MS than in the non-MS subgroup (Table 1).

As expected, serum concentrations of total cholesterol, triglycerides, glucose and insulin and HOMA-IR value were greater, and concentration of HDL-cholesterol was lower in the subgroup with MS than in the non-MS subgroup (Table 2).

We did not observe any significant difference in plasma concentrations of visfatin, TNF-α or sTNFRs between the MS and the non-MS subgroup. In the MS subgroup, but not in the non-MS subgroup, visfatin levels were significantly higher than in the controls. There were no differences in plasma concentrations of TNF-α and sTNFR2 between the MS and non-MS subgroups and controls. Only plasma sTNFR1 levels were greater in the control group than in the non-MS one, but not greater than in the MS subgroup (Table 3).

Correlations between visfatin and other study parameters

In the combined analysis of obese subgroups and controls, we observed significant positive correlations between age (R = 0.32, p = 0.01) or TNF-α levels (R = 0.21, p = 0.03) and visfatin levels (Figs. 1, 2). In the separate subgroup analysis, such correlations were statistically significant only in the most numerous MS subgroup (R = 0.33, p = 0.01; R = 0.31, p = 0.01, respectively). Additionally, in the MS subgroup we found a significant negative correlation between absolute values of free fat mass and visfatin levels (R = –0.30, p = 0.01).

Moreover, in the MS subgroup, visfatin levels were significantly related to serum concentrations of triglycerides (R = 0.45, p = 0.04), insulin (R = 0.53, p = 0.01) and HOMA index value (R = 0.53, p = 0.01).

In all study subgroups, visfatin level correlated with body mass, BMI, waist circumference, body fat mass and serum glucose levels.

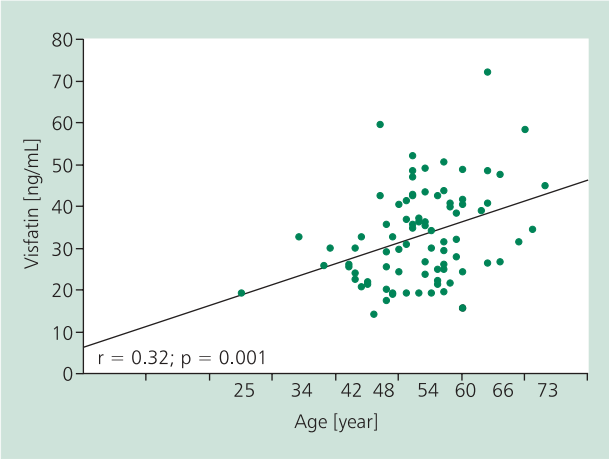

Figure 1. Correlation between age and visfatin level in the combined analysis of obese women fulfilling or not the criteria of metabolic syndrome and controls

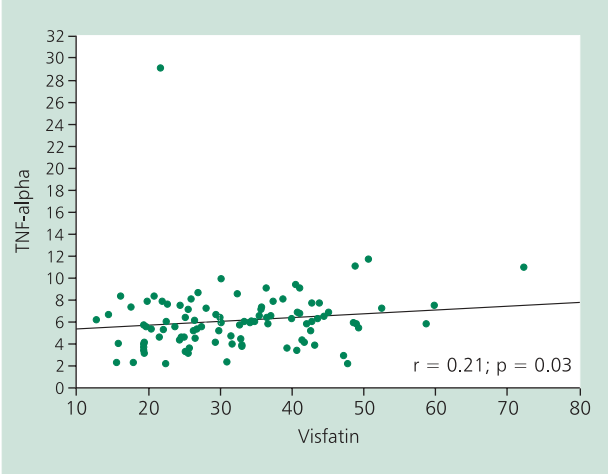

Figure 2. Correlation between visfatin and TNF-alpha levels in the combined analysis of obese women fulfilling or not the criteria of metabolic syndrome and controls

DISCUSSION

Visfatin and TNF-α are produced in visceral adipose tissue and their production is increased in cases of obesity [6, 9, 12]. Elevated circulating levels of visfatin and TNF-α in obese subjects have also been previously reported [19–21]. However, in our study we did not observe differences in TNF-α levels between obese subjects with and without MS. Visfatin levels in MS were only slightly and non-significantly higher than in non-MS obese subjects, but were significantly higher than in the controls. It should be stressed that the obese patients were significantly older, and in part, the higher visfatin values observed may be due to this. This thesis is supported by the significant positive correlation between age and visfatin levels that was found in the combined analysis of all study subgroups. On the other hand, despite the fact that the non-MS subgroup was also older than the control group, visfatin levels were similar.

In contrast to the results obtained by Berndt et al. [12], who reported significant correlations between visfatin levels and BMI and the percentage of body fat in a wide-ranging group of obese subjects, we did not find corresponding univariate correlation with body mass, BMI, waist circumference and percentage of body fat in the present study nor in our previously published study [21]. The contribution of adipose tissue to the amount of circulating visfatin seems to be complex. The expression of visfatin in subcutaneous tissue in lean subjects outweighs visceral secretion [22], while in obesity this proportion is reversed, and visceral secretion becomes predominant. The issue of the influence of body mass on plasma visfatin level is even more complicated. Recently published experimental studies both in chickens and humans have reported the expression of visfatin in skeletal muscles [22, 23]. This certainly may disturb the correlation between body fat mass and plasma visfatin concentration. Interestingly, in the present study we observed a negative correlation between fat free mass and visfatin levels in the MS subgroup. This finding is difficult to explain based on the present knowledge.

The proinflammatory properties of visfatin have also been reported. Dahl et al. [24] demonstrated that visfatin is highly expressed by lipid-loaded macrophages in atherosclerotic lesions, particularly in plaques of symptomatic patients, while Moshen et al. [25] showed that recombinant visfatin activates human leukocytes and induces cytokine production. In $CD14^+$ monocytes, visfatin enhanced the production of IL-1β, TNF-α, and especially of IL-6. It has also been reported that the expression of visfatin in macrophages infiltrating visceral white adipose tissue is higher than in mature adipocytes [26]. Our findings of positive correlations between visfatin and TNF-α levels in MS subjects further document its proinflammatory action. Recently, de Luis et al. [27] revealed that TNF-α is independently related to plasma visfatin levels in patients with impaired fasting glucose.

An important finding is the positive correlation between plasma visfatin concentrations and insulin levels or HOMA-IR values in MS subjects. Such a correlation does not support the previously reported insulin-sensitising properties of visfatin. Unfortunately, our data do not explain the underlying mechanisms. In experimental conditions in healthy lean volunteers, hyperglycaemia induced by glucose infusion was followed by an increase in visfatin concentration. The increase was related to glucose elevation, and prevented by co-administration of insulin [28]. In our previous study we described positive correlations between serum glucose and plasma visfatin levels only in lean women, whereas we found in obese women (without concomitant diseases) a positive interrelation between serum insulin and plasma visfatin levels [21]. The results obtained by Brema et al. [29] confirm the elevation of plasma visfatin concentration in insulin resistance conditions in patients with early onset of type 2 diabetes

mellitus. In this study, plasma visfatin levels were reduced after aerobic exercise, which typically improves insulin sensitivity and decreases chronic inflammation associated with obesity. We can only hypothesise that increased visfatin levels in hyperinsulinaemia and/or insulin resistance conditions stimulate the production of TNF-α, and in turn exaggerate insulin resistance.

Therefore, it seems that the role of visfatin in obesity, insulin resistance and MS is complex and is yet to be completely elucidated. Further studies are necessary to clarify the role of visfatin in the pathogenesis of obesity and insulin resistance.

Limitations of the study

The main limitation of our study is the lack of direct visceral fat assessment and differences in age between study subgroups and controls. However, the presented results do bring into question the beneficial role of visfatin.

CONCLUSIONS

The present study suggests that visfatin in MS should be regarded as a proinflammatory factor indirectly favouring the development of insulin resistance.

Conflict of interest: *none declared*

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Wisfatyna i czynnik martwicy nowotworów alfa (TNF-α) w zespole metabolicznym

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Streszczenie

Wstęp: W badaniach doświadczalnych wykazano, że czynnik martwicy nowotworów alfa (TNF-α) oraz interleukina 6 (IL-6) zmniejszają ekspresję genu wisfatyny w adipocytach. Stwierdzono także, że wisfatyna stymuluje produkcję cytokin w leukocytach i monocytach.

Cel: Celem pracy była ocena związków między stężeniem w osoczu wisfatyny i TNF-α oraz jego rozpuszczalnych receptorów u otyłych kobiet z zespołem metabolicznym i bez niego.

Metody: Do badania włączono 92 otyłe kobiety. Na podstawie kryteriów IDF z 2005 r. zespół metaboliczny zdiagnozowano u 71 osób (wiek 53 ± 9 lat; wskaźnik masy ciała 39,1 ± 5,6 kg/m^2, obwód talii 109,6 ± 11,4 cm), natomiast 21 badanych nie spełniało kryteriów rozpoznania tego schorzenia (wiek 52 ± 9 lat, wskaźnik masy ciała 36,3 ± 5,2 kg/m^2, obwód talii 104,7 ± 11,0 cm). Grupę kontrolną stanowiło 14 zdrowych kobiet o prawidłowej masie ciała. U wszystkich badanych poza pomiarami antropometrycznymi oceniono skład ciała metodą bioimpedancji. Stężenie w osoczu wisfatyny, TNF-α i rozpuszczalnych receptorów TNF oznaczono metodą ELISA. Insulinooporność oceniono metodą pośrednią za pomocą HOMA-IR.

Wyniki: W podgrupie z zespołem metabolicznym, ale nie w podgrupie otyłych bez tego zespołu, stężenie w osoczu wisfatyny było istotnie wyższe niż w grupie kontrolnej. Nie zaobserwowano różnic stężeń w osoczu wisfatyny, TNF-α i sTNFRs między podgrupami z zespołem metabolicznym i bez niego. Tylko w podgrupie z zespołem metabolicznym oraz w analizie połączonej całej grupy badanej i kontrolnej zanotowano istotną dodatnią korelację między stężeniem w osoczu wisfatyny i TNF-α (odpowiednio $R = 0,31$; $p = 0,01$; $R = 0,21$; $p = 0,03$). Dodatkowo w podgrupie z zespołem metabolicznym występowała istotna dodatnia korelacja między stężeniem w osoczu wisfatyny a wartościami HOMA-IR ($R = 0,53$; $p = 0,01$).

Wnioski: Uzyskane wyniki sugerują że wisfatyna w zespole metabolicznym może działać jako czynnik prozapalny, niezależnie sprzyjający rozwojowi insulinooporności.

Słowa kluczowe: wisfatyna, TNF-α, sTNFRs, otyłość, zespół metaboliczny

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