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Original article

ADIPONECTIN AND INSULIN RESISTANCE IN PRE-DIABETES AND EARLY TYPE 2 DIABETES MELLITUS

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Abstract

Background: The aim of our study was to evaluate adiponectin level in subjects with pre-diabetes and to compare it with the levels in newly diagnosed type 2 diabetes and healthy subjects. We also studied associations of adiponectin level with other metabolic indices.

Methods and methods: study included 48 individuals with pre-diabetes (IGT), 33 newly diagnosed type 2 diabetes mellitus (DM) subjects, and 68 healthy subjects. Blood samples were analysed for fasting blood glucose, insulin, $HbA1_C$ and adiponectin. According to BMI control subjects were separated into obese (BMI ≥ 27) and non-obese (BMI < 27) groups, and according to HOMA-IR they were divided into insulin-sensitive (HOMA-IR < 3), and insulin-resistant (HOMA-IR > 3) subsets.

Results: median serum adiponectin levels were not statistically significantly different in IGT and DM groups (7.56 (4.18; 13.57) vs. 7.63 (5.04; 11.73), p = 0.858). Serum adiponectin was significantly lower in IGT group as compared to obese-control group (7.56 (4.18; 13.57) vs. 14.37 (7.68; 19.24), p = 0.004). Comparison of obese and non-obese control groups showed that serum adiponectin level was lower in obese subjects, but the difference was not statistically significant (14.37 (7.68; 19.24) vs. 17.41 (9.95; 24.57) μg/ml p = 0.181), whereas insulin-resistant controls had significantly lower adiponectin levels when compared to insulin-sensitive ones (11.85 (8.31; 16.72) vs. 18.69 (10.72; 28.32) μg/ml p = 0.04). Adiponectin showed inverse correlation with HOMA-IR.

Conclusions: adiponectin level was similar in IGT and type 2 diabetes groups and was significantly lower than in control subjects.

Keywords:
adiponectin, insulin resistance, prediabetes, type 2 diabetes

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Introduction

Obesity is a well-known risk factor for type 2 diabetes, and about 90% of the humans affected by this disease are overweight or obese [1]. The pathophysiology linking obesity to type 2 diabetes is not completely understood,

but adipokines are thought to be involved [2]. Adiponectin is a recently discovered protein that seems to be exclusively secreted by adipocytes and is the most abundant adipose tissue–derived protein [3, 4]. In contrast to other adipokines (such as leptin, interleukin-6 and others) that are often elevated in obese subjects, adiponectin is reduced [5–7].

In animal studies, adiponectin has been shown to have insulin-sensitizing properties. Administration of adiponectin reversed insulin resistance in various mouse models of obesity and diabetes [8]. Injections of recombinant adiponectin protein into mice acutely lowered plasma fatty acids and glucose, as well as improved insulin sensitivity [9]. Chronic administration of the recombinant adiponectin in mice also reduced body weight [10]. In animal studies adiponectin increased insulin action via effects on hepatic glucose production [8] and by increasing fat oxidation and lowering circulating free fatty acids [10–12].

Lower plasma levels of adiponectin relative to the normal controls were documented in human subjects with obesity [4], insulin resistance [5, 13] and type 2 diabetes [6] in several cross-sectional studies. Results from a few prospective studies suggest that low adiponectin level is predictive of insulin resistance or diabetes [14–17]. Although the association of low adiponectin concentration and type 2 diabetes is rather well studied, only few studies investigated adiponectin level in prediabetes (e.g. impaired glucose tolerance and impaired fasting glucose) [18, 19]. The results of these studies are rather conflicting, one group showing similar levels of adiponectin in pre-diabetes and type 2 diabetes [19] and the other one reporting significantly higher levels of adiponectin in pre-diabetes as compared to type 2 diabetes [18]. It seems that association of adiponectin with insulin resistance occurs early in obesity development, however it remains unclear if this association is of further importance in prediabetes and early stages of type 2 diabetes and remains the same through all glucose intolerance development stages.

The objective of our study was to evaluate adiponectin level in subjects with pre-diabetes and to compare it with the levels in newly diagnosed type 2 diabetes and healthy (normal glucose tolerance) subjects. We also studied associations of adiponectin level with insulin resistance in all study subjects and in separate groups.

Research design and methods

The present study included 48 individuals with prediabetes (impaired glucose tolerance or impaired fasting glucose (IGT)), 33 newly diagnosed type 2 diabetes mellitus subjects (DM), and 68 healthy subjects.

Lithuanian Bioethics Committee approved study protocol, and all subjects signed an informed consent form after being informed on the purpose and procedures of the study.

Subjects were evaluated at Vilnius University Faculty of Medicine, Vilnius University Hospital "Santariskiu Klinikos" Endocrinology centre and Laboratory Medicine centre from September 2005 until May 2007. The evaluation involved a full medical history and physical examination, including anthropometric measurements (weight, height, waist and hip circumferences, total body fat mass and percentage measured by bioelectrical impedance (OMRON BF 302 body fat monitor), arterial blood pressure and pulse rate. Subjects were excluded if they had a known history of cardiovascular disease, stroke or transient ischemic attack, uncontrolled hypertension, or any other serious chronic disease requiring active treatment.

Metabolic studies

Venous blood sample were taken in the morning after 12 hours of fasting. With the subject in the sitting position, an intravenous needle was inserted into a forearm vein. Blood samples were drawn for fasting blood glucose, insulin and adiponectin. Standard OGTT was performed for all study subjects: 75 grams of oral glucose load over a 2-minute period was given, and blood samples were obtained again 2 hours after for plasma glucose measurement. Based on the fasting serum glucose and OGTT, categories of glucose tolerance status were defined by WHO 1998 recommendations, and study subjects were divided into impaired glucose tolerance or impaired fasting glucose (IGT), type 2 diabetes mellitus (DM) and control (C) groups. According to BMI control subjects were separated into obese (BMI $\geq$ 27) (Obese-C) and non-obese (BMI < 27) (Non-obese-C) groups.

Glucose and insulin were determined using standard laboratory methods. Serum adiponectin was measured with the radioimmunoassay (RIA) kits following the manufacturer's protocols (Linco Research Inc., St. Louis, MO).

Calculations and Statistical analyses

The body mass index (BMI) was calculated as weight (kilograms) divided by height squared (meters). The homeostasis model assessment of insulin resistance (HOMA-IR), an index of insulin resistance, was calculated using an equation as described [21].

Data were summarized using standard procedures. Descriptive statistics are presented as mean ± SD or median (quartiles) for normally and non-normally distributed parameters respectively. Student's t test was used to analyse data with normal distribution, whereas Mann and Whitney U-test was applied to compare non-parametric parameters. Spearman's correlation coefficient was calculated to explore correlation between adiponectin and other variables. SPSS 15.0 for Windows software (SPSS, Chicago, IL) was used for statistical analysis. A P value < 0.05 was considered statistically significant.

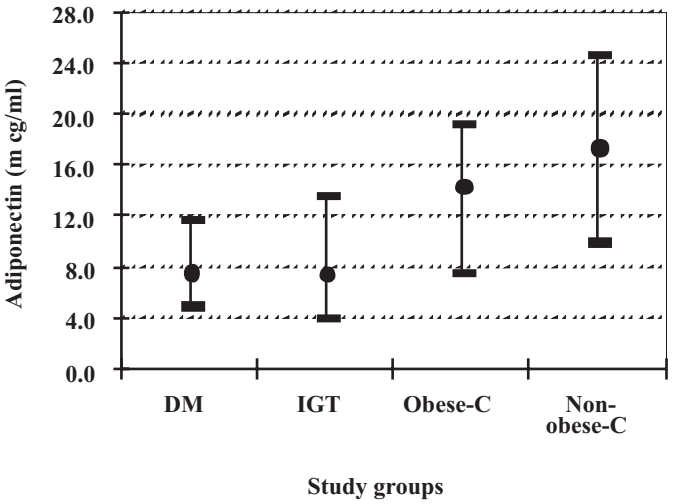

Fig. 1. Serum adiponectin in IGT, DM, Obese-Control and Non-obese-Control subjects
Dots show median; error bars show quartiles.

Results

Demographic and clinical characteristics of all study groups are presented in Table 1.

As shown in the Table 1, the subjects from IGT and DM groups were older, more often suffered from arterial hypertension whereas more controls, particularly non-obese ones, were smoking.

Effects of glucose metabolism on serum adiponectin levels

In our study we have compared anthropometric and biochemical parameters in IGT and DM groups. As shown in Table 2, anthropometric parameters (BMI, waist, hip, waist to hip ratio (W/H), body fat mass and fat percentage) did not statistically significantly differed in IGT and DM groups, whereas systolic and diastolic blood pressure and heart rate were significantly higher in DM subjects. Despite the fact that fasting blood glucose and HOMA-IR were significantly higher in DM group, adiponectin (7.56 (4.18; 13.57) vs. 7.63 (5.04; 11.73) µg/ml, p = 0.858) (Figure 1).

To assess further the influence of glucose metabolism status on adiponectin levels, we compared IGT and Obese-C groups, which had similar anthropometric and clinical characteristics (Table 2). IGT and Obese-C groups were comparable with regard to anthropometric measures (BMI, waist, hip, fat mass and percentage); they also had similar blood pressure and heart rate. Fasting blood glucose was significantly higher in IGT group, but plasma insulin and HOMA-IR did not significantly differ. The analysis showed that adiponectin was significantly lower in IGT group than in Obese-C group (7.56 (4.18; 13.57) vs. 14.37 (7.68; 19.24), p = 0.004) (Figure 1).

Effects of obesity on adiponectin levels in subjects with normal glucose tolerance

To evaluate the influence of obesity on adiponectin

Table 1. Baseline demographic and clinical characteristics of study groups

Parameters	IGT	DM	Obese-C	Non-obese-C
n	48	33	44	24
Age (years)	53.67 ± 9.28	55.82 ± 8.45	48.14 ± 9.32	47.87 ± 9.06
Sex (F/M)	20/13	30/18	31/13	16/8
Hypertension (n)	41 (85.4%)	30 (90.9%)	28 (63.6%)	9 (37.5%)
Smoking (n)	9 (18.8%)	8 (24.2%)	12 (27.3%)	7 (29.2%)
Systolic BP (mmHg)	146.34 ± 20.96	159.35 ± 24.69	142.32 ± 23.63	134.00± 21.93
Diastolic BP (mmHg)	90.94 ± 12.45	95.77 ± 12.11	89.59 ± 12.92	86.13 ± 13.03

Table 2. Anthropometric and biochemical variables of IGT, DM and Obese-C groups.

Parameters	IGT	DM	P1	Obese-C	P2
BMI (kg/m2)	33.31 ± 5.38	35.28 ± 6.71	0.150	32.34 ± 4.62	0.360
Waist (cm)	105.31 ± 12.08	110.38 ± 12.83	0.077	100.74 ± 11.72	0.071
Hip (cm)	113.74 ± 10.00	117.38 ± 12.91	0.236	113.95 ± 9.03	0.857
W/H	0.93 ± 0.06	0.94 ± 0.08	0.498	0.88 ± 0.07	0.003
Fat mass (kg)	34.68 ± 10.02	35.41 ± 9.53	0.755	33.81 ± 9.57	0.678
Fat mass (%)	36.61 ± 7.15	37.13 ± 7.56	0.764	36.2 ± 7.10	0.789
Systolic BP (mmHg)	146.34 ± 20.96	159.35 ± 24.69	0.013	142.32 ± 23.63	0.311
Diastolic BP (mmHg)	90.94 ± 12.45	95.77 ± 12.11	0.038	89.59 ± 12.92	0.419
Heart rate (beats/min)	69.94 ± 8.04	76.35 ± 8.12	0.001	69.89 ± 7.92	0.879
Fasting blood glucose (mmol/l)	6.29 (6.05; 6.57)	8.00 (7.3; 11.4)	<0.0001	5.30 (5.11; 5.70)	<0.0001
Insulin (μU/ml)	12.05 (8.24; 20.53)	15.30 (10.55; 21.65)	0.226	12.30 (7.65; 18.81)	0.525
HOMA-IR	3.48 (2.25; 5.84)	5.80 (3.75; 9.11)	<0.0001	3.17 (1.86; 4.20)	0.093
Adiponectin (μg/ml)	7.56 (4.18; 13.57)	7.63 (5.04; 11.73)	0.858	14.37 (7.68; 19.24)	0.004

P^1 for pairwise comparisons, IGT versus DM;

P^2 for pairwise comparisons, IGT versus Obese-C.

concentration we compared Obese-C and Non-obese-C subjects. As shown in Table 3, Obese-C subjects had significantly higher BMI, waist and hip circumferences, larger amount of body fat mass and percentage; they also had higher systolic and diastolic blood pressure. Comparison of the metabolic parameters showed that fasting blood glucose was quite similar in these study groups, whereas insulin and HOMA-IR were significantly higher in Obese-C subjects. Serum adiponectin concentration was lower in Obese-C than in Non-obese-C group (14.37 (7.68; 19.24) vs. 17.41 (9.95; 24.57) μg/ml), but the difference was not statistically significant (p = 0.181) (Figure 1).

To asses adiponectin levels in whole group of investigated subjects and the influence of insulin resistance, we have empirically divided subjects into 3 groups: insulin-sensitive group (HOMA-IR ≤ 3), intermediate insulin-sensitivity group (HOMA-IR 3–6) and insulin resistant group (HOMA-IR ≥ 6). The analysis of two extremes (HOMA-IR ≤ 3 and HOMA-IR ≥ 6) showed that there is

Table 3. Anthropometric and biochemical characteristics of Obese-Control and Non-obese-Control groups

Parameters	Obese-Control	Non-obese-Control	*p*
BMI (kg/m^2)	32.34 ± 4.62	23.55 ± 1.93	*<0.0001*
Waist (cm)	100.74 ± 11.72	83.17 ± 7.85	*<0.0001*
Hip (cm)	113.95 ± 9.03	98.90 ± 3.90	*<0.0001*
W/H	0.88 ± 0.07	0.84 ± 0.06	*0.004*
Fat mass (kg)	33.81 ± 9.57	18.26 ± 5.30	*<0.0001*
Fat mass (%)	36.2 ± 7.10	26.64 ± 7.44	*<0.0001*
Systolic BP (mmHg)	142.32 ± 23.63	134.00± 21.93	0.210
Diastolic BP (mmHg)	89.59 ± 12.92	86.13 ± 13.03	0.406
Fasting blood glucose (mmol/l)	5.30 (5.11; 5.70)	5.18 (4.81; 5.54)	0.333
Insulin (μU/ml)	12.30 (7.65; 18.81)	6.45 (5.15; 9.58)	*<0.0001*
HOMA-IR	3.17 (1.86; 4.20)	1.53 (1.14; 2.23)	*<0.0001*
Adiponectin (μg/ml)	14.37 (7.68; 19.24)	17.41 (9.95; 24.57)	0.181

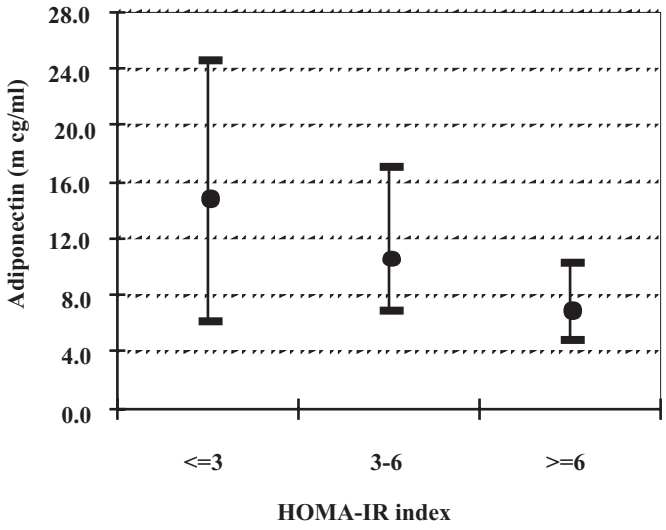

Fig. 2. Serum adiponectin in all subjects with different insulin resistance levels.
Dots show median; error bars show quartiles

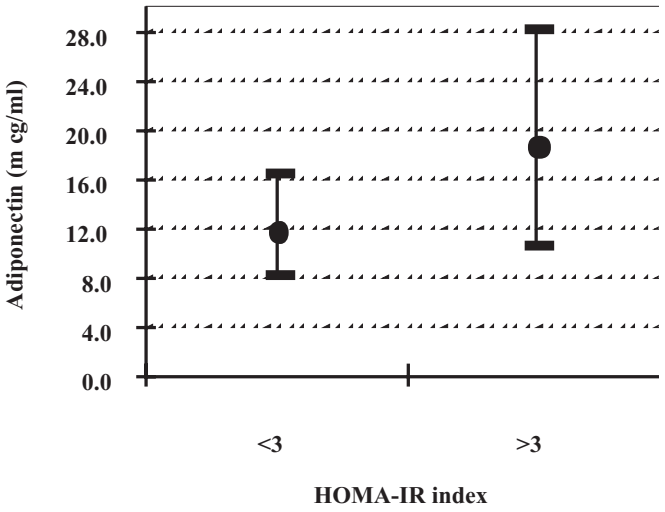

Fig. 3. Serum adiponectin in insulin-resistant and insulin-sensitive control subjects.
Dots show median; error bars show quartiles.

Table 4. Spearman's rank correlation of adiponectin with metabolic and anthropometric variables among study participants; all subjects combined and study groups.

Parameter	**All subjects**	**IGT**	**DM**	**Obese-C**	**Non-obese-C**
BMI (kg/m^2)	-.279(*)	-.108	-.359	-.120	-.342
Waist (cm)	-.447(**)	-.366(*)	-.508(**)	-.274	-.649(**)
Hip (cm)	-.205(*)	-.069	-.202	-.062	-.374
W/H	-.509(**)	-.487(**)	-.345	-.371(*)	-.642(**)
Fat mass (kg)	-.251(**)	-.143	-.434(*)	-.102	-.034
Fat mass (%)	-.033	-.017	.029	.038	.155
Systolic BP (mmHg)	-.148	-.048	.268	-.233	.033
Diastolic BP (mmHg)	-.110	.116	.020	-.207	-.109
Fasting blood glucose (mmol/l)	-.221(**)	.226	-.150	.113	-.082
Insulin (μU/ml)	-.316(**)	-.247	-.253	-.336(*)	-.156
HOMA-IR	-.340(**)	-.375(**)	-.285	-.340(*)	-.191

** Correlation is significant at the 0.05 level (2-tailed).*
*** Correlation is significant at the 0.01 level (2-tailed).*

clear transition from higher adiponectin level and better insulin sensitivity to lower adiponectin level and higher insulin-resistance state (Figure 2). Adiponectin level was more then twice lower in the group with the highest insulin resistance when compared with the insulin-sensitive group (7.14 (4.49; 10.29) μg/ml vs. 14.92 (6.29; 24.60) μg/ml; $p < 0.0001$).

To asses further adiponectin levels in subjects with normal glucose tolerance and the influence of insulin resistance, we have empirically divided control subjects into insulin-resistant (HOMA-IR > 3) and insulin-sensitive (HOMA-IR < 3). The analysis of our results showed that insulin-resistant controls had significantly lower adiponectin levels when compared to insulin-sensitive ones (11.85 (8.31; 16.72) vs. 18.69 (10.72; 28.32) μg/ml $p = 0.04$) (Figure 3), but when compared to IGT group insulin-resistant control subjects had higher levels of adiponectin (7.56 (4.18; 13.57) vs. 11.85 (8.31; 16.72) μg/ml, $p = 0.040$).

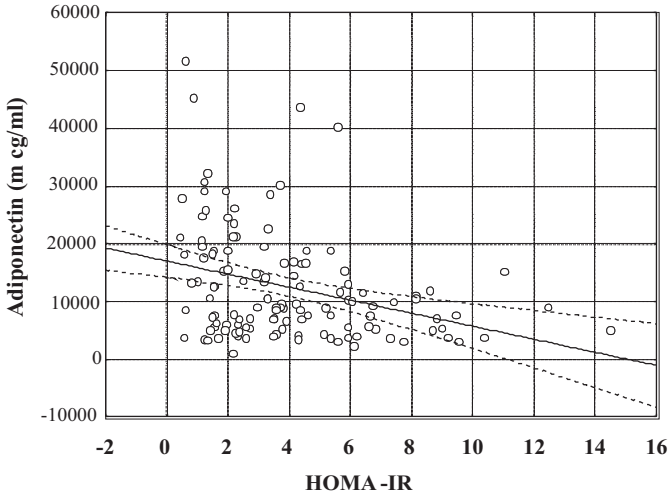

Fig. 4. Correlation of adiponectin with HOMA-IR in all subjects combined.

Relationship of adiponectin levels with metabolic and obesity parameters

Among all study subjects, adiponectin showed strongest inverse correlations with waist circumference, W/H, and HOMA-IR, whereas more modest, but statistically significant negative correlations were observed between adiponectin and BMI, hip circumference, body fat mass, fasting glucose and insulin (Table 4.).

Correlation analysis in study groups (Table 4.) showed that adiponectin did not significantly correlate with indices of overall obesity (BMI and body fat mass or percentage body fat), but negative associations with indices of visceral obesity (waist circumference and W/H) were rather strong in all study groups. Adiponectin significantly correlated with W/H ($r = -.487$, $p < 0.0001$) in IGT group and with waist circumference ($r = -.508$, $p = 0.004$) in DM subjects. Inverse correlation was observed between adiponectin and HOMA-IR in all subjects combined, although individual variations probably had some influence on final data in different study groups (Figure 4). In Obese-C group adiponectin showed the inverse correlations with HOMA-IR, insulin and W/H. In Non-obese-C subjects adiponectin significantly inversely correlated with waist ($r = -.649$, $p < 0.0001$) and W/H ($r = -.642$, $p < 0.0001$).

Discussion

In this study we demonstrated that adiponectin level was similar in IGT and type 2 diabetes groups and in both these groups it was significantly lower when compared with obese control group. On the other hand, adiponectin was not statistically significantly different in obese control when compared with non-obese control subjects.

Previous studies have found that plasma adiponectin levels in IGT were both lower [18] and similar [19] when compared with type 2 diabetes subjects. Osei K. et al. [18] studied first degree relatives of African-American patients and showed that adiponectin levels were significantly lower in the type 2 diabetes group, whereas in IGT and normal glucose tolerance subjects adiponectin levels were significantly higher. Although it was not the primary endpoint of the study, Bluher M et al. [19] reported similar levels of total plasma adiponectin in IGT and type 2 diabetes Caucasian subjects, and in both groups adiponectin levels were lower than in subjects with normal glucose tolerance. Possibly due to the same racial group studied and similar study groups characteristics the results of our study confirmed the findings of Bluher et al. [19]. The results of our study suggest that adiponectin secretion is already altered in pre-diabetic conditions, and as the majority of subjects with IGT eventually will develop type 2 diabetes, based on the results of our study we speculate that transition from pre-diabetes to type 2 diabetes is not associated with further alterations in adiponectin metabolism. Our data also suggest that metabolic alterations seen in IGT state are strong enough to further lead to diabetes development.

Adiponectin level was found to be decreased in obese subjects in several previous studies [4, 22]. In our investigation obese subjects with normal glucose tolerance had lower adiponectin levels than non-obese ones, but the difference did not reach statistical significance, probably due to rather small number of non-obese control subjects studied.

The results of our study confirm that adiponectin levels are strongly associated with visceral obesity and insulin resistance [5, 13, 22]. Indeed, we demonstrated that adiponectin significantly inversely correlated with waist circumference and waist-to-hip ratio, but not with indices of overall obesity: BMI and body fat mass.

We also found that insulin-resistant glucose tolerant subjects had significantly lower adiponectin level than insulin-sensitive ones, but statistically significantly higher than in IGT and DM subjects.

In summary, our study demonstrated that adiponectin levels are reduced in pre-diabetic conditions to the same extend as in early type 2 diabetes, therefore we suggest that adiponectin secretion is more closely related to insulin resistance than to simply transition from pre-diabetes

to diabetes which was not associated with further alteration in adiponectin secretion. We conclude that insulin sensitivity and visceral obesity rather than obesity per se appears to be the major determinants of serum adiponectin levels.

References

1. Ardern CI, Janssen I, Ross R, Katzmarzyk PT. Development of health-related waist circumference thresholds within BMI categories. Obes Res. 2004; 12: 1094–103.
2. Jazet IM, Pijl H, Meinders AE. Adipose tissue as an endocrine organ: impact on insulin resistance. Neth J Med. 2003; 61: 194–212.
3. Trujillo ME, Scherer PE. Adiponectin: journey from an adipocyte secretory protein to biomarker of the metabolic syndrome. J Intern Med. 2005; 257: 167–75.
4. Arita Y, Kihara S, Ouchi N et al. Paradoxical decrease of an adipose-specific protein, adiponectin, in obesity. Biochem Biophys Res Commun. 1999; 257: 79–83.
5. Weyer C, Funahashi T, Tanaka S, et al. Hypoadiponectinemia in obesity and type 2 diabetes: close association with insulin resistance and hyperinsulinemia. J Clin Endocrinol Metab. 2001; 86: 1930–5.
6. Hotta K, Funahashi T, Arita Y et al. Plasma concentrations of a novel, adipose-specific protein, adiponectin, in type 2 diabetic patients. Arterioscler Thromb Vasc Biol. 2000; 20: 1595–9.
7. Cnop M, Havel PJ, Utzschneider KM et al. Relationship of adiponectin to body fat distribution, insulin sensitivity and plasma lipoproteins: evidence for independent roles of age and sex. Diabetologia. 2003; 46: 459–69.
8. Berg AH, Combs TP, Du X, Brownlee M, Scherer PE. The adipocyte-secreted protein Acrp30 enhances hepatic insulin action. Nat Med. 2001; 7: 947–53.
9. Yamauchi, T, Kamon, J, Waki, H, et al. The fat-derived hormone adiponectin reverses insulin resistance associated with both lipoatrophy and obesity. Nat Med. 2001; 7: 941–6.
10. Fruebis, J, Tsao, TS, Javorschi, S et al. Proteolytic cleavage product of 30-kDa adipocyte complement-related protein increases fatty acid oxidation in muscle and causes weight loss in mice Proc Natl Acad Sci USA. 2001; 98: 2005–10.
11. Combs TP, Berg AH, Obici S, Scherer PE, Rossetti L. Endogenous glucose production is inhibited by the adipose-derived protein Acrp30. J Clin Invest. 2001; 108: 1875–81.
12. Yamauchi T, Kamon J, Minokoshi Y at al. Adiponectin stimulates glucose utilization and fatty-acid oxidation by activating AMP-activated protein kinase. Nat Med. 2002; 8: 1288–95.
13. Yamamoto Y, Hirose H, Saito I et al. Correlation of the adipocyte-derived protein adiponectin with insulin resistance index and serum high-density lipoprotein-cholesterol, independent of body mass index, in the Japanese population. Clin Sci. 2002; 103: 137–42.
14. Lindsay RS, Funahashi T, Hanson RL et al. Adiponectin and development of type 2 diabetes in the Pima Indian population. Lancet. 2002; 360: 57–8.
15. Snehalatha C, Mukesh B, Simon M, Viswanathan V, Haffner SM, Ramachandran A. Plasma adiponectin is an independent predictor of type 2 diabetes in Asian Indians. Diabetes Care. 2003; 26: 3226–9.
16. Daimon M, Oizumi T, Saitoh T et al. Decreased serum levels of adiponectin are a risk factor for the progression to type 2 diabetes in the Japanese population: the Funagata Study. Diabetes Care. 2003; 26: 2015–20.
17. Snijder MB, Heine RJ, Seidell JC et al. Associations of adiponectin levels with incident impaired glucose metabolism and type 2 diabetes in older men and women: the Hoorn Study. Diabetes Care. 2006; 29: 2498–503.
18. Osei K, Gaillard T, Schuster D. Plasma adiponectin levels in high risk African-Americans with normal glucose tolerance, impaired glucose tolerance, and type 2 diabetes. Obesity Research. 2005; 13: 179–85.
19. Bluher M, Brennan AM, Kelesidis T et al. Total and high-molecular weight adiponectin in relation to metabolic variables at baseline and in response to an exercise treatment programm. Diabetes Care. 2007; 30: 280–5.
20. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabet Med. 1998; 15(7): 539–53.
21. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia. 1985; 28: 412–9.
22. Wei-Siung Y, Wei-Jei L, Funahashi T et al. Plasma adiponectin levels in overweight and obese Asians. Obes Res. 2002; 10: 1104–10.

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ADIPONEKTINAS IR REZISTENTIŠKUMAS INSULINUI PREDIABETO IR ANKSTYVAIS 2 TIPO CUKRINIO DIABETO FORMŲ ATVEJAIS

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Santrauka

Įvadas. Buvo tiriama adiponektino koncentracija ir adiponektino ryšys su gliukozės apykaita ir rezistentiškumu insulinui asmenims esant prediabetui bei pradinėms 2 tipo cukrinio diabeto stadijoms.

Tiriamieji asmenys ir metodai. Buvo ištirti 48 asmenys, kuriems buvo diagnozuotas prediabetas (gliukozės tolerancijos sutrikimas), 33 asmenys, kuriems buvo naujai diagnozuotas 2 tipo cukrinis diabetas ir 68 sveiki asmenys. Priklausomai nuo kūno masės indekso (KMI) sveikų asmenų grupė buvo padalinta į nutukusių (KMI ≥ 27) ir nenutukusių (KMI < 27) bei priklausomai nuo insulino rezistentiškumo indekso į jautrių insulinui (HOMA-IR < 3) ir rezistentiškų insulinui (HOMA-IR > 3) grupes.

Rezultatai. Adiponektino koncentracijos prediabeto ir diabeto grupėse statistiškai nesiskyrė (7,56 (4,18; 13,57) ir 7,63 (5,04; 11,73) μg/ml, p = 0,858). Serumo adiponektino koncentracija, nežiūrint antropometrinių ir kitų klinikinių panašumų, buvo statistiškai patikimai mažesnė asmenims su gliukozės tolerancijos sutrikimu, palyginus su sveikų nutukusių asmenų grupe (7,56 (4,18; 13,57) ir 14,37 (7,68; 19,24), p = 0,004). Nutukusių sveikų asmenų adiponektino koncentracija buvo mažesnė palyginus nenutukusių sveikų asmenų grupe, tačiau šis skirtumas nebuvo statistiškai patikimas (14,37 (7,68; 19,24) ir 17,41 (9,95; 24,57) μg/ml p = 0,181), kai tuo tarpu insulinui rezistentiški sveiki asmenys turėjo statistiškai patikimai mažesnę adiponektino koncentraciją palyginus su jautrių insulinui asmenų grupe (11,85 (8,31; 16,72) ir 18,69 (10,72; 28,32) μg/ml p = 0,04). Adiponektino koncentracija koreliavo neigiamai su rezistentiškumo insulinui HOMA-IR indeksu.

Išvados. Adiponektino koncentracijos buvo panašios GTS ir 2 tipo cukrinio diabeto grupėse, tačiau buvo statistiškai patikimai mažesnės palyginus su sveikų asmenų grupe.

Raktažodžiai:

adiponektinas, rezistentiškumas insulinui, prediabetas, 2 tipo cukrinis diabetas