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Concentrations of omentin and vaspin versus insulin resistance in obese individuals

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ABSTRACT

Introduction: Omentin and vaspin are adipokines manifesting a potentially protective action against obesity-associated metabolic disturbances.
Aim: Evaluation of relationship between serum concentrations of omentin and vaspin on one hand and indices of insulin resistance and anthropometric parameters in obese individuals on the other.
Material and methods: The studies were conducted on 64 individuals. The investigated group (37 obese patients) included the subgroup with normal glucose tolerance (NGT) and with abnormal glucose tolerance (AGT). The control group (n = 27) included healthy individuals with normal body weight. In all participants anthropometric analyses and biochemical tests, including estimation of omentin and vaspin concentrations were performed, and insulin resistance by HOMA-IR was evaluated.
Results: Concentrations of examined adipokines manifested no significant differences between the examined groups. Median values of the index defining ratio between studied adipokine and degree of insulin resistance, i.e. omentin/HOMA-IR, proved to be different in the investigated and the control group while no such difference could be noted in cases of vaspin/HOMA-IR indices. In the studied population a negative relationship was detected between serum concentration of omentin and systolic blood pressure ($p < 0.04$). Values of omentin/HOMA-IR index manifested a correlation with values of most anthropometric parameters ($p < 0.0001$), blood pressure ($p < 0.0001$) concentrations of TG ($p < 000.1$) and HDL ($p < 0.0001$), ISI_{basal} ($p < 0.00001$), ISI_{gly} ($p < 0.0001$), Quicki ($p < 0.00001$) and fasting insulinaemia ($p < 0.00001$). In the case of vaspin/HOMA-IR index only its positive relationship with HDL concentration was noted ($p < 0.05$).
Conclusion: In context of date of correlation, multiple regression and values of area of under receiver operating characteristics curve omentin, as compared to vaspin, seems to provide a better predictor of insulin resistance in obese individuals.

1. Introduction

Chronically positive energy equilibrium, low physical activity and genetic predisposition lead to hypertrophy and hyperplasia of adipocytes. This abnormality, termed the adiposopathy or "sick fat" unfavourably affects the paracrine, endocrine function and immune response of adipose tissue [1]. The relationship between amounts of adipose tissue in the body and secretion of individual adipokines is significant because these molecules differ between each other in their function. A disturbed homeostasis of their secretion, results in a disturbed metabolism, accompanying an excessive body weight, including insulin resistance which, next to chronic inflammation of low intensity, is thought to represent the main cause of diabetes type 2 development, of cardiovascular diseases and metabolic syndrome [2,3].

In analyses a relationship between an increased secretion of well recognised adipokines, such as leptin and resistin and a reduced production of adiponectin and development of insulin resistance was detected [4,5]. The noticed correlations prompted attempts to define similar relationships related to the newly recognised adipokines, i.e. to omentin and vaspin. Functions of these two adipokines have not been till now sufficiently documented.

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Performed investigations indicate in vitro and in animal experimental models show that the discussed molecules may favourably influence glucose metabolism and sensitivity of tissues to action of insulin. Analyses on the concentrations of omentin and vaspin, depending on the degree of tissue in insulin sensitivity in obese people is relatively few and their results are not conclusive. Taking the above into account, this study aimed at identification and evaluation of the relationship between concentration of omentin and vaspin in serum on one hand and indices of insulin resistance and anthropometric parameters in obese individuals on the other.

2. Material and methods

The project was accepted by the local bioethical commission (decision No. 431/13 of 9th May, 2013). The investigated group included 37 obese individuals (17 women and 20 men). The qualified patients were subdivided into two groups, differing in the result of 75 g Oral Glucose Tolerance Test (OGTT) according to World Health Organization (WHO) and International Diabetes Federation [6]:

- Obese individuals with glycaemia in 120th min of OGTT $<$7.8 mmol/L – NGT (Normal Glucose Tolerance),
- Obese individuals with glycaemia in 120th of OGTT $\geq$7.8 mmol/L – AGT (Abnormal Glucose Tolerance).

Exclusion criteria involved earlier diagnosed and treated diabetes mellitus, impaired glucose tolerance, impaired fasting glucose and moreover chronic diseases of endocrine glands, liver, kidneys and pancreas, neoplastic diseases and cardiovascular incidents. None of the participants cultivated record-seeking sports. The control group included 27 healthy individuals (16 women and 11 men) with normal body weight. In both groups age of participants ranged between 30 and 60 years. Detailed characteristics of the groups are presented in Table 1.

In the morning in all participants anthropometric examination was conducted. Body height and weight of fasting participants in underwear only were estimated using a certified electronic scale (SECA I360 Wireless, Hamburg, Germany). Moreover, circumferences were measured in waist and in hips in line with recommendations of WHO [7]. The results permitted to calculate anthropometric indices: BMI (Body Mass Index), WHpR (Waist-Hip Ratio) and WHtR (Waist-Height Ratio). Composition of the body were examined using electric bioimpedance (1500 MDD, Bodystat, Isle of Man, United Kingdom).

Resting seated blood using a digital electronic tensiometer (model 705IT, Omron Corporation, Kyoto, Japan) pressure was measured three times and an average value was calculated according to guidelines of European Society of Hypertension and European Society of Cardiology [8]. Subsequently blood was sampled from cubital vein for biochemical estimations.

Fasting glycaemia and lipid profile (triglyceridaemia – TG, high density lipoprotein – HDL) were estimated using standardised commercial methods. Concentration of low density lipoprotein – LDL was calculated using the formula of LDL = TC – (HDL + TG/5). For estimation of insulinaemia an immunochemical test was used with application of Chemiflex test protocols (Abbott Diagnostics, Illinois, U.S.A). Fasting concentrations of insulin and glucose were used to calculate insulin resistance in tissues, HOMA-IR (Homeostasis Model Assessment of Insulin Resistance), according formula: HOMA-IR = fasting glycaemia (mmol/L) $\times$ fasting insulinaemia (mU/L)/22.5 [9]. Additionally ISI_{basal} (insulin sensitivity index) according formula: 10^4/(fasting insulinaemia (μU/mL) $\times$ fasting glycaemia (mg/dL)), ISI_{gly} insulin sensitivity index for glycaemia according formula: 2/[(fasting insulinaemia (μU/mL)/12$\times$ (fasting glycaemia (mg/dL)/100) +1] [10,11], Quicki (quantitative insulin sensitivity check index) according formula: 1/(log(fasting insulinaemia μU/mL) + log(fasting glycaemia mg/dL)) [10], TyG (the triglycerides $\times$ glucose index) according formula: Ln[fasting triglyceridaemia (mg/dL) $\times$ fasting glycaemia (mg/dL)/2] [12] and VAI (visceral adiposity index) according formula: (waist circumference

Table 1
Mean values ± standard deviations of selected parameters in investigated group and control group.

PARAMETER	INVESTIGATED GROUP OBESITY n = 37			CONTROL GROUP NORMAL BODY WEIGHT n = 27	p**
	Normal glucose tolerance (NGT) n = 17	Abnormal glucose tolerance (AGT) n = 20	p*		
Body weight (kg)	113.9 ± 24.6	119.8 ± 27.6	ns	69.4 ± 14.2	<0.0001
BMI (kg/m^2)	36.8 ± 5.7	41.1 ± 6.4	<0.04	23.8 ± 2.8	<0.0001
Waist circumference (cm)	117.5 ± 15.0	123.9 ± 15.3	ns	80.1 ± 9.8	<0.0001
Hips circumference (cm)	121.5 ± 14.7	123.6 ± 10.9	ns	98.0 ± 6.1	<0.0001
WHpR	1.0 ± 0.1	1.0 ± 0.1	ns	0.8 ± 0.1	<0.0001
WHtR	0.7 ± 0.1	0.7 ± 0.1	<0.02	0.5 ± 0.0	<0.0001
Content of adipose tissue (%)	43.4 ± 7.6	46.3 ± 7.0	ns	23.4 ± 4.7	<0.0001
Lean body mass (%)	56.6 ± 7.6	53.7 ± 7.0	ns	76.6 ± 4.7	<0.0001
Fasting glycaemia (mmol/L)	4.9 ± 0.6	6.0 ± 0.9	<0.0005	4.9 ± 0.4	<0.0002
Glycaemia at 120'of OGTT (mmol/L)	5.5 ± 1.2	10.0 ± 1.7	<0.0001	–	–
Insulinaemia (μU/mL)	11.1 ± 6.8	16.2 ± 7.6	<0.02	6.0 ± 2.6	<0.0001
HOMA-IR	2.5 ± 1.5	4.5 ± 2.7	<0.003	1.3 ± 0.6	<0.0001
Triglyceridaemia (mmol/L)	2.4 ± 1.3	2.3 ± 1.1	ns	1.0 ± 0.5	<0.0001
HDL concentration (mmol/L)	1.2 ± 0.3	1.1 ± 0.3	ns	1.9 ± 0.5	<0.0001
LDL concentration (mmol/L)	3.8 ± 1.0	3.3 ± 1.5	ns	3.2 ± 0.7	ns
Systolic blood pressure (mm Hg)	141.0 ± 16.0	145.3 ± 18.6	ns	120.4 ± 17.4	<0.0001
Diastolic blood pressure (mm Hg)	88.8 ± 8.3	92.9 ± 9.8	ns	80.0 ± 9.3	<0.0001
Omentin concentration (nmol/L)	71.28 ± 27.75	63.89 ± 23.19	ns	72.53 ± 28.67	ns
Vaspin concentration (nmol/L)	0.12 ± 0.12	0.18 ± 0.16	ns	0.13 ± 0.15	ns
Omentin/HOMA-IR	**34.98 ± 20.94**	**19.30 ± 14.75**	**<0.01**	**66.74 ± 42.46**	**<0.0001**
Vaspin/HOMA-IR	0.08 ± 0.15	0.05 ± 0.05	ns	0.12 ± 0.16	ns

n—number of people; p*—level of statistical significance upon comparison of NGT vs AGT; p**—level of statistical significance upon comparison between investigated group and control group; ns—statistically insignificant difference; BMI—Body Mass Index; WHpR—Waist-Hip Ratio; WHtR—Waist-Height Ratio, HOMA-IR—Homeostasis Model Assessment of Insulin Resistance, HDL—high density lipoprotein level, LDL—low density lipoprotein level.