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Obesity Endocrinology Congress 2019: Association between omentin and chemerin levels and their changes within one year in non-morbid overweight and obese adults

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Obesity is typically related to diabetes mellitus type 2, although it has become a serious problem even among type 1 diabetics. Diabetes mellitus type 1 is a chronic disease characterised by absolute lack of insulin, resulting from autoimmune destruction of the pancreatic β-cells. This malfunction interferes not only in carbohydrate metabolism but also in that of fat and protein, and leads to a wide range of multisystem abnormalities with a common etiopathogenesis of formidable macro- and microvascular disease.

In obese diabetics, redundant fat tissue is associated with insulin resistance, dyslipidemia, hypertension, endothelial dysfunction and a pro-inflammatory state. Obesity can thus markedly contribute to the development of diabetic complications. In these effects adipose tissue-secreted cytokines/adipokines are implicated.

Omentin-1 (intelectin-1, intestinal lactoferrin receptor, endothelial lectin HL-1, galactofuranose-binding lectin), a 313-amino acid peptide, is an anti-inflammatory adipokine preferentially expressed in stromal vascular cells of visceral adipose tissue. It is suggested that this substance makes an important contribution to the physiological difference between visceral and subcutaneous adipose tissue. It is abundant also in human vasculature, the small intestine, colon, thymus and heart. Omentin-1 is the major circulating form; it also has a homologue designated as omentin-2 and their genes are localised adjacent to each other at 1q22-q23 in the region linked to diabetes mellitus type 2. Both omentin homologues in circulating form correlate with expression in visceral fat tissue. Omentin-1, as with adiponectin, can activate 5′-AMP-activated protein kinase and endothelial nitric oxide synthase. Via this activation, omentin-1 is essentially involved in cellular energy homoeostasis and vascular tone regulation. Omentin-1 increases insulin signal transduction, enhances insulin-stimulated glucose transport in human adipocytes (but has no effect on basal glucose uptake) and contributes to regulation of lipid metabolism. In contrast to adiponectin, omentin-1 can inhibit activation of JNK (c-Jun N-terminal kinases), and thus it is suggested that omentin-1 is involved in stress responses, expression of heat shock proteins, T-cell differentiation and apoptosis.

Omentin-1 is known to modulate immune reactions of the organism, and thus may have an anti-inflammatory effect, a significant association with inflammatory markers has been reported. It takes part in defence mechanisms by binding to galactofuranoses on bacteria. Omentin-1 inhibits the TNF-α mediated induction of pro-inflammatory molecules in vascular endothelial cells. Important roles have also been suggested in vasodilatation, development of endothelial dysfunction and arterial calcification. Plasma levels of omentin-1 are decreased in insulin-resistant and pro-inflammatory states (diabetes mellitus type 1 and 2, obesity, polycystic ovary syndrome and so on). Obesity and insulin-resistant states are associated with enhanced endogenous cholesterol synthesis as well, cholesterol absorption remains lower in comparison with non-insulin-resistant population. In obese patients plasma omentin-1 levels increase after weight loss; however, dynamics of plasma omentin-1 and markers of cholesterol metabolism in obese type 1 diabetics have not been characterized so far. Possible insulin-sensitising and anti-inflammatory effect of omentin-1 could have a positive role in changes of cholesterol metabolism in diabetes mellitus type 1.

About half of EU adult population is overweight, including 16% being obese (21% in Latvia). Early recognition and monitoring of individuals that are at high risk of developing diabetes and cardiovascular diseases is essential. Positive correlation of serum chemerin and negative correlation of omentin with weight, lipids and insulin resistance indicators has been described; however, information on inter-relation between changes in these parameters is scarce, especially in non-morbid overweight and obese adults.

Objective: To determine the association between chemerin and omentin levels at baseline and their changes in clinically healthy overweight and obese individuals within a year.

Materials & Methods: We used data from our randomised controlled study with 123 clinically healthy individuals with a BMI above 25 m2 /kg in the age group of 30 to 45: (47% men, age 36,8±4,2 years, BMI 32,0±4,3 kg/m2 ; total cholesterol 5,4±0,9 mmol/L; HDL-cholesterol 1,4±0,3 mmol/L; fasting glucosae 5,2±0,5 mmol/L; HOMA-IR 3,1±1,7; 46% metabolically unhealthy according to metabolic syndrome definition; 32% smokers; 38% diagnosed with liver steatosis on CT scan). All participants received a consultation for lifestyle changes to support weight loss. All group showed slight weight and waist circumference decrease after 1 year. Biochemical parameters (lipids, fasting glucose and insulin) and cytokines (omentin, chemerin) were assessed at baseline and after 1 year using Spearman's correlation test.

Results: We found a weak positive correlation between chemerin and omentin (rs=0.295; p=0.001) at baseline,

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contradicting our expectations. Multiple linear regression adjusted by age and gender retained significant relationship between omentin and chemerin (B=0.088; 95% CI 0.033, 0.143; p=0.002). After one year there was a weak positive correlation of omentin changes with chemerin changes (rs=0.186; p=0.042). However, multiple linear regression adjusted by age and gender showed no association between omentin and chemerin changes. Conclusion: A positive rather than negative relationship between chemerin and omentin in non-morbid overweight and obese adults imply that other factors besides anthropometric and metabolic indicators might be affecting omentin and chemerin levels in this group.

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