Retinol-binding protein 4 expression in subcutaneous neck adipose tissue and serum in patients with and without metabolic syndrome

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OBJECTIVES

Retinol-binding protein 4 (RBP4) is considered as an important mediator of insulin resistance and metabolic syndrome (MS). In adipose tissue it is mainly secreted from visceral depots. The residual metabolic risk which remains beyond that caused by visceral adipose tissue seems to be induced by upper body subcutaneous adipose tissue. This large pathogenic depot might be represented by neck subcutaneous adipose tissue.

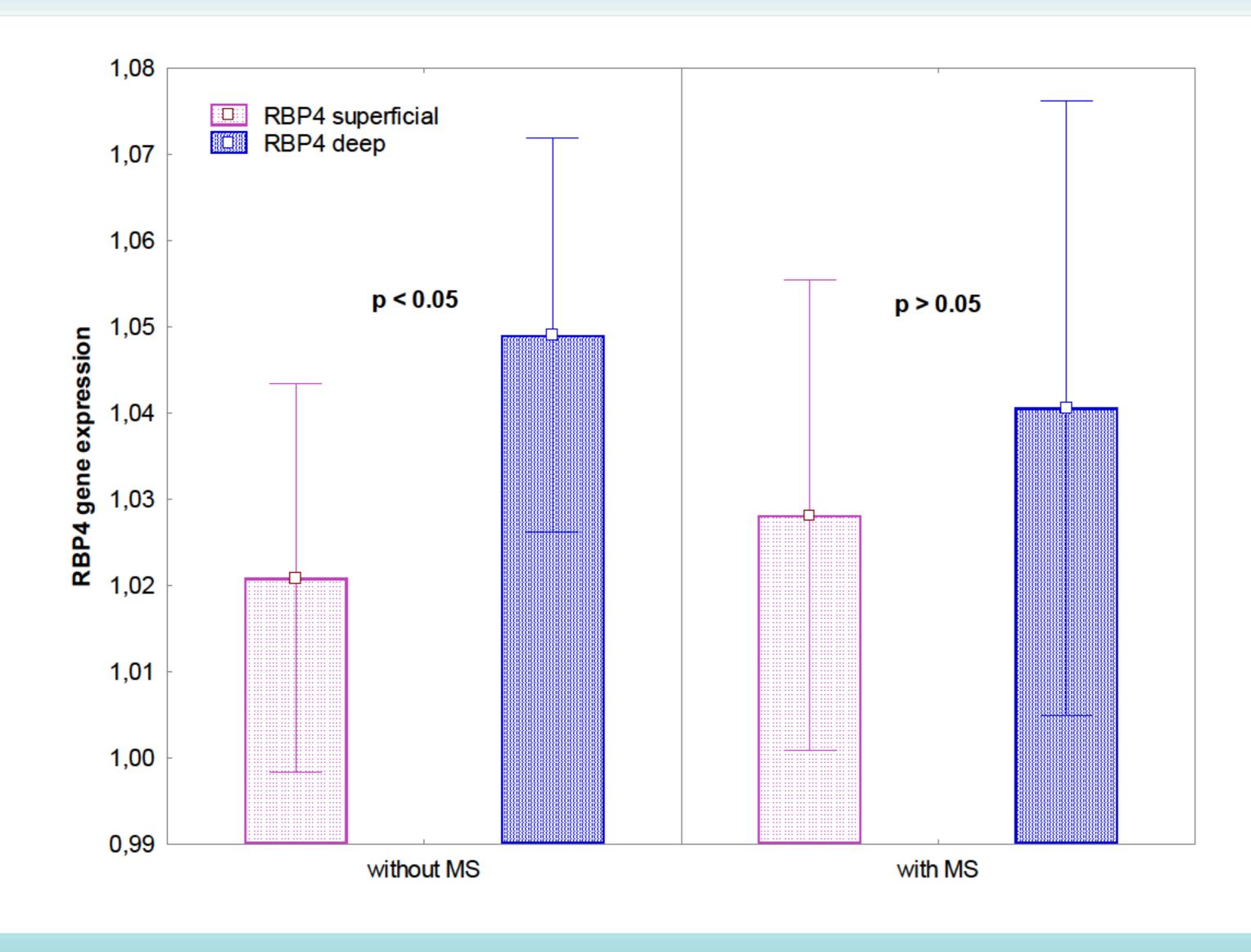
The aim of this study was to examine and to compare RBP4 gene expressions in paired superficial and deep neck subcutaneous adipose tissue. We also investigated their associations with metabolic risk factors and serum RBP4.

METHODS

Samples of serum, superficial and deep neck subcutaneous adipose tissue were taken in 38 patients during routine thyroid or vascular neck surgery. Serum was taken preoperatively to determine insulin, glucose, triglycerides, HDL-cholesterol, C-reactive protein and RBP4. Anthropometric measurements and bioelectric impedance analysis were also performed. Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated and anthropometric measurements like body weight and height, body mass index, waist and neck circumferences were performed. RBP4 gene expression in both adipose tissue samples were analysed by RQ-PCR method.

Study participants were divided in two groups - with and without MS according to revised NCEP ATP III criteria. Study participants did not have any inflammatory or malignant diseases. They also did not have any thyroid or other specific neck mass that could particularly enlarge neck circumference.

RESULTS



RBP4 gene expressions in adipose tissue samples and serum level were not different between the group with and without metabolic syndrome.

RBP4 gene expressions were significantly lower in superficial than in deep adipose tissue in the group without MS. In patients with MS different RBP4 gene expressions between layers of adipose tissue were not present.

In the whole sample of participants superficial RBP4 gene expression positively correlated with fat mass, insulin and HOMA index.

Serum RBP4 concentration did not correlate with RBP4 gene expressions in any of the analyzed adipose tissue depots samples. Serum RBP4 concentration correlated with waist circumference, insulin, HOMA index and triglycerides.

CONCLUSIONS

Serum RBP4 concentration in our study supported its association with insulin resistance, but there was no difference in serum RBP4 between patients with and without metabolic syndrome. That is probably due to a number of known demographic and other confounders in small studies. The results of our study particularly suggest differences in RBP4 gene expression between layers of neck subcutaneous adipose tissue in patients without metabolic syndrome. The functional differences of distinct anatomical layers are well known features of abdominal subcutaneous adipose tissue. Lower RBP4 gene expression in superficial layer of neck subcutaneous adipose tissue seems to follow a lower inflammatory adipokine spectrum of superficial abdominal subcutaneous adipose tissue. Our results also suggests that metabolically more favorable lower RBP4 gene expression in superficial layer of neck subcutaneous adipose tissue disappear in subjects with MS. With MS dysfunctional changes seem to predominate in all depots and all layers of upper body subcutaneous adipose tissue which might emphasize its contribution to insulin resistance and other adverse metabolic developments.

References

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