**BETA3-ADRENOCEPTOR GENE EXPRESSION IN RAT ADIPOSE TISSUE IN RESPONSE TO REPEATED IMMUNE CHALLENGE**

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Next to beta1- and beta2-adrenoceptors, the beta3-adrenoceptors represent important regulatory sites to control cardiac function. Beta3-adrenergic receptors in the heart may protect the myocardium against adverse effects of excessive catecholamine stimulation. This may be particularly important under demanding situations, such as stress and immune system activation. A typical location of beta3-adrenergic receptors is the adipose tissue and they are involved in the regulation of lipolysis.

The aim of these studies was to verify the hypothesis that stress associated with repeated immune challenge has a negative impact on the gene expression of beta3-adrenoceptors and related regulatory factors in the adipose tissue.

Female and male Sprague-Dawley rats were intraperitoneally treated either with vehicle or lipopolysaccharide (LPS) in increasing doses for 5 days (50-200 microg/kg). Two hours after the last injection, the retroperitoneal adipose tissue and selected brain regions were collected. Concentrations of selected mRNAs were measured using real-time PCR.

Immune system activation by repeated treatment with LPS was confirmed by increased mRNA levels of interleukin-6. This immune challenge resulted in a decrease in gene expression of beta3-adrenoceptors in the adipose tissue. Adipogenic factor (peroxisome proliferator-activated receptor gamma, PPAR-gamma) gene expression was decreased after administration of LPS. As to the adipokines, both adiponectin and leptin mRNA levels decreased significantly. Treatment with LPS failed to modify expression of resistin, however mRNA levels of these adipose tissue-specific secretory factor were significantly higher in males compared to females. In contrast to adipose tissue, gene expression of beta3-adrenoceptors was unchanged in the hippocampus and prefrontal cortex.In conclusion, parallel decreases in gene expression of beta3-adrenoceptors, PPAR-gamma, leptin and adiponectin encourage further research on possible role of beta3-adrenoceptors in the adipogenesis and control of the formation of adipokines in white adipose tissue in response to immune challenge. Supported by grants of VEGA 2/0128/14 and APVV-14-0840.