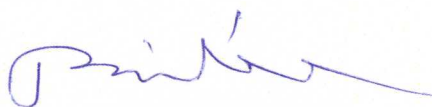


THIS IS TO CERTIFY THAT

Balázs Pósfai

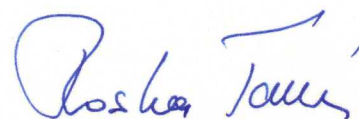
**has attended the From Medicine to Bionics
Ph.D. Conference
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FROM MEDICINE TO BIONICS

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HUNGARY'S RENEWAL



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NEW SZÉCHENYI PLAN

issue (WAT) there is an increasing interest on the relationship between BAT and obesity. Both BAT and WAT contains macrophages, which have an impact on body metabolic and inflammatory functions. Fractalkine(FKN) is a chemokine that attracts macrophages into several tissues, including adipose tissues. FKN receptor (Cx3CR1) is expressed mainly by macrophages. The aim was to reveal the role of macrophages in BAT function in obesity. We used transgenic mice wherein Cx3CR1 was replaced by gfp reporter gene. +/gfp mice have functioning Cx3CR1 while gfp/gfp mice do not express Cx3CR1. Mice were fed with that enriched diet (FatED) for 10 weeks. +/gfp FatED - but not gfp/gfp FatED mice – gained more weight and had higher relative BAT weight than normal diet (ND) fed mice. Lipid droplets of brown adipose cells were larger in FatED than ND groups. Rt-PCR analysis showed elevated FKN, MCP-1, GFP, IL-6, TNFa, Arg1, UCP1, PPARG, NPY and Nucleob2 mRNA levels in BAT in response to FatED. In case of TNFa, MCP-1, PPARG and Nucleob2 gfp/gfp FatED mice had lower levels of mRNA than +/gfp FatED mice.

These results reveal increased number of macrophages in BAT of obese mice and FKN signaling plays a role in the traffic and accumulation of these cells. Similarly with our previous results, mRNA levels of inflammatory cytokines tend to be lower in gfp/gfp FatED mice than +/gfp FatED mice. We revealed that BAT is capable to express NPY, which might affect polarization of tissue specific macrophages, and Nucleob2, which might play a role in the transmission of anorexigenic signal from the periphery to the brain during obesity.

Positive effects of N-acetyl cysteine in the animal model of schizophrenia

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Schizophrenia is a mental disorder, which affects nearly 1% of the population. Polymorphisms at G72 locus are considered to show one of the most robust associations with the disease. Transgenic mice that express the human gene G72 show behavioural changes that are expected in an animal model of schizophrenia. Physiological data suggest changes in the plasticity of perforant path synapses on granule cells of the dentate gyrus in these animals, therefore, we investigated whether there are any morphological changes behind those changes seen in physiological measurements. Using both pre- and post-embedding methods, we examined these excitatory synapses of stratum moleculare with electron microscope and we also carried out stereological measurements using both electron and light microscopy. We found, that synapses are at least 33% larger in transgenic (TG, n=5) mice compared to wild type (n=5) littermates, and contain around 2 times more NMDA receptors, while total number of excitatory synapses is reduced in

stratum moleculare of transgenic mice. The reason of these observed changes could be a G72-protein induced mitochondrial alteration, or a change in the concentration of the NMDA receptor co-agonist D-serine.

Because N-acetyl-cysteine (NAC)-treatment ameliorated behavioural alterations in G72-TG animals, we tested whether this treatment would rescue the abovementioned changes, as well. Our preliminary data suggest that several weeks of treatment may indeed reverse observed changes in these synapses.

The effect of certain steroid hormones on the expression of genes involved in the metabolism of free radicals

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Background and aims: Steroid hormones have influence on the antioxidant processes of cells. However, the molecular mechanism of this effect is not clear. Our aim was to examine how steroid hormones influence the expression of certain genes that play a role in processes connected with free radicals.

Methods: Blood was obtained from ten healthy volunteers. Neutrophil granulocytes were separated and treated with either 17-β-estradiol, progesterone, testosterone or cortisol in a concentration of 10-8M for 2 hours. Whole RNA was isolated and reverse transcription was carried out in treated and control groups. Relative quantification was performed with SYBR Green assay and gene-specific oligonucleotides.

Results: The expression of CuZn and extracellular superoxide dismutase (SOD) was not altered by any of the steroid hormones. On the other hand, Mn SOD expression was doubled by cortisol and progesterone, and elevated three- and fourfold by 17-β-estradiol and testosterone, respectively. Myeloperoxidase expression was almost doubled by cortisol and progesterone, while neither of the other steroids had any effect. The expression of NADPH oxidase was decreased by progesterone.

Conclusions: According to our results we conclude that the antioxidant effect of steroid hormones is at least in part (but not entirely) carried out through transcriptional regulation of certain enzymes. Subsequent studies are needed to examine the non-genomic, membrane receptor mediated effect of steroids on antioxidant processes.