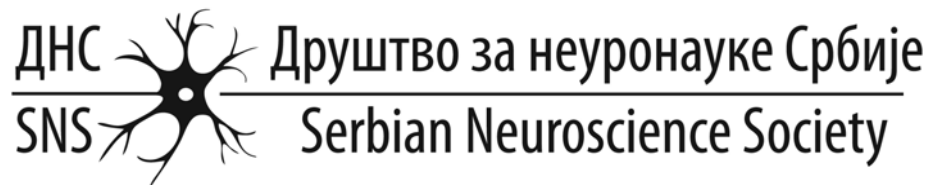




7th CONGRESS OF SERBIAN NEUROSCIENCE SOCIETY
with international participation

BOOK OF ABSTRACTS

Belgrade
October 25-27, 2017.



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Department of Biochemistry, Institute for Biological Research "Siniša Stanković", University of Belgrade, Belgrade, Serbia

Introduction. The macrophage migration inhibitory factor (MIF) is involved in the regulation of glucose metabolism in the hippocampus, while fructose overconsumption has been associated with changes in hippocampal insulin sensitivity and synaptic plasticity, resulting in disturbances in learning and memory. Therefore, we hypothesized that MIF deficiency in combination with fructose diet (FD) may affect insulin sensitivity and hippocampal synaptic plasticity, thus leading to behavioral changes. **Methods.** The effects of 9-week 20% FD on energy intake and insulin sensitivity in wild type (WT) and MIF deficient (MIF^{-/-}) C57Bl/6J male mice were analyzed. Inhibitory Ser³⁰⁷ phosphorylation of insulin receptor substrate 1 (IRS-1) was used as hallmark of hippocampal insulin resistance. Novel object recognition test (NOR) was used to assess exploratory behavior. Synaptic plasticity was estimated by polysialylated-neural cell adhesion molecule (PSA-NCAM) protein level. **Results.** WT and MIF^{-/-} mice on fructose diet had increased energy intake, while systemic insulin sensitivity was disturbed in all MIF^{-/-} mice. Hippocampal pIRS-1 Ser³⁰⁷ was elevated in all animals compared to WT on standard diet (SD). MIF^{-/-} animals on SD showed impaired recognition memory in NOR. Finally, although PSA-NCAM was decreased in WT animals on FD and MIF^{-/-} on SD, its level in fructose-fed MIF^{-/-} mice was the same as in the WT on SD. **Conclusion.** These preliminary results show that both MIF deficiency and fructose caloric overload are implicated in impairment of systemic and hippocampal insulin sensitivity. However, behavioral changes observed in MIF^{-/-} mice were normalized after fructose feeding, which coincided with the changes in synaptosomal PSA-NCAM protein level.

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