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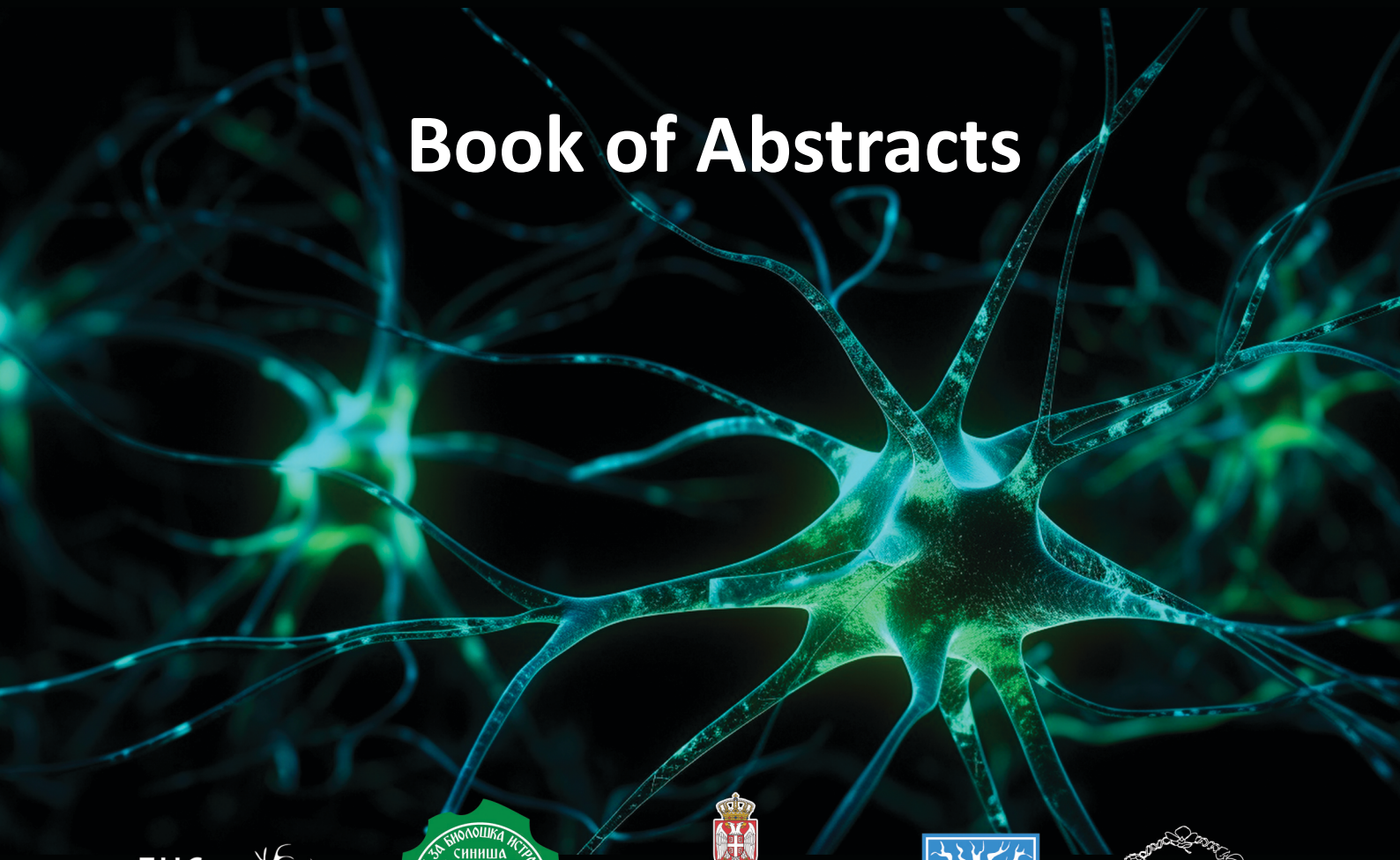

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Upregulation of glial markers with absence of a typical proinflammatory profile in the hippocampus of A53T mice as a model of Parkinson's disease

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Parkinson's disease (PD) is characterized by both motor and non-motor symptoms. Current research indicates that neuroinflammation, along with activated glial cells, represents one of the key factors in the pathogenesis of PD. The aim of this study was to assess non-motor symptoms of PD as well as the expression of gene markers of microgliosis, astrogliosis and neuroinflammation in a transgenic A53T mouse model, which is recommended for studying PD and other synucleinopathies. These transgenic mice express an A53T missense mutant form of human alpha-synuclein under the control of the murine prion promoter.

Depression-like symptoms and anhedonia were assessed using Tail Suspension Test and Sucrose Preference Test respectively, while memory deficits were assessed using Novel Object Recognition Test along with Object Relocation Test. Gene expression of ionized calcium-binding adapter molecule (Iba-1), glial fibrillary acidic protein (GFAP α and δ form) and cytokines associated with the microglial pro-inflammatory phenotype (TNFα, IL-1β, IL-6) was assessed using RT-PCR. Brain regions of interest were the prefrontal cortex, hippocampus, and striatum of 6 months old A53T male mice. Non-transgenic littermates were used as controls. Obtained results suggest that A53T mice compared to non-transgenic littermates exert 1) the presence of memory deficits and anhedonia, 2) no difference in the expression of glial activity markers and pro-inflammatory cytokines in the cortex and striatum, and 3) increased expression of glial activity markers in the hippocampus, but without significant changes in the expression of cytokines. Overall, better understanding of the role of active glia in the model is needed.