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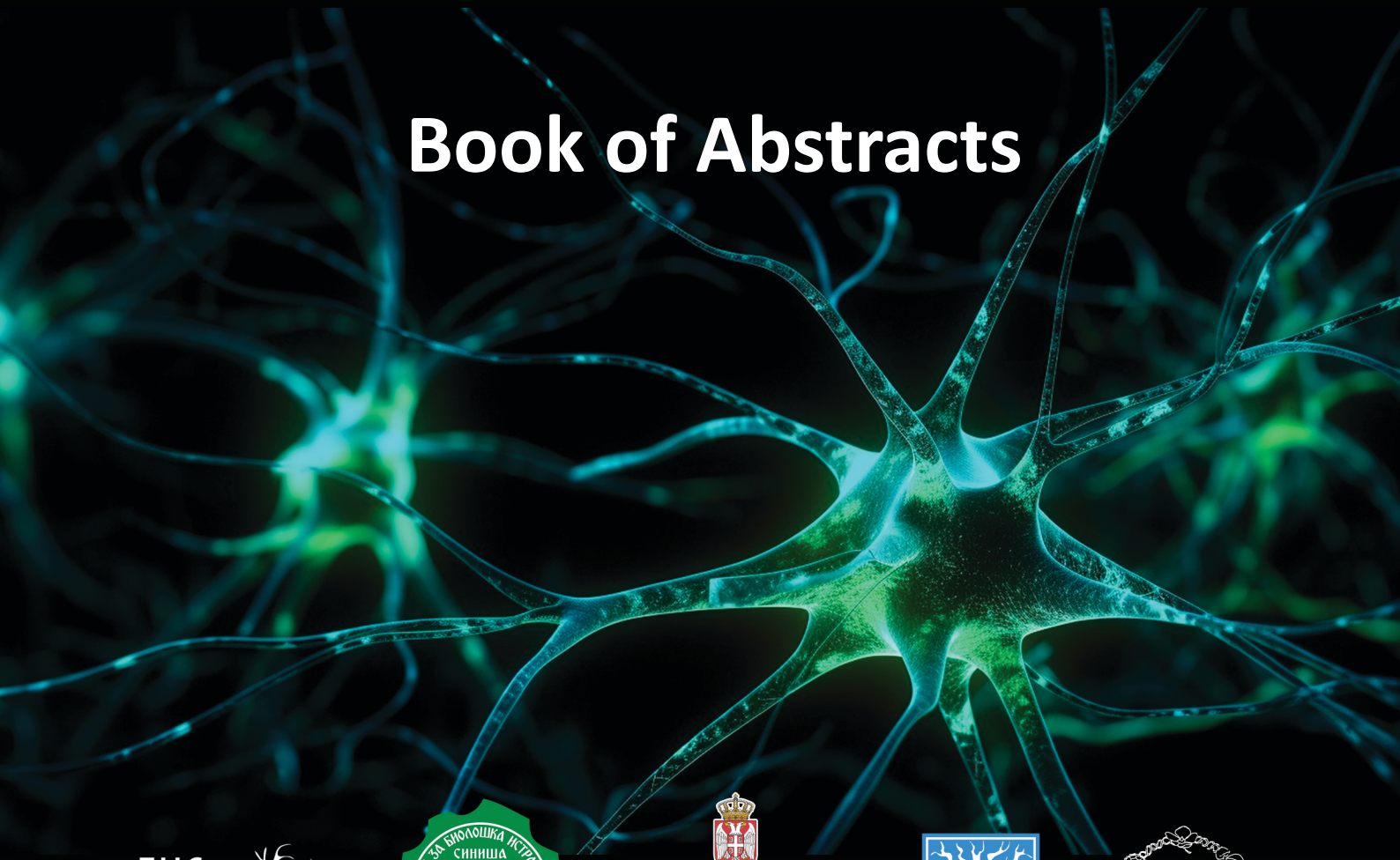

Book of Abstracts

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GABAergic parvalbumin-expressing interneurons play a role in memory impairment in rat models of Parkinson's disease

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We investigated the role of hippocampal GABAergic parvalbumin-expressing (PV) interneurons in spatial and hippocampus-dependent memory abilities in rat models of Parkinson's disease (PD).

Experiments were performed in adult male Wistar rats, including physiological controls (n=14) and toxin lesion-induced PD models: PD cholinopathy (n=10), hemiparkinsonism (n=7), and hemiparkinsonism with PD cholinopathy (n=6). Behavioral assessments and PV immunohistochemistry were performed 14 and 42 days after lesions. Spatial habituation test and novel object recognition test were used to assess spatial and hippocampus-dependent short- and long-term recognition memory.

All experimental groups had no motor impairments during the follow-up period ($X^2 \geq 2.01$, $p \geq 0.07$). Although hippocampal PV expression remained unchanged over time in PD cholinopathy ($z \geq -1.91$, $p \geq 0.06$), we evidenced impairments in spatial, short- and long-term recognition memory, but only at day 42 ($X^2 \geq 0.38$, $p=0.83$; $t=0.13$, $p=0.91$). In the hemiparkinsonian rats, unchanged hippocampal PV expression ($z \geq -1.52$, $p \geq 0.14$) was followed by impairment in spatial memory ($X^2 \geq 2.87$, $p \geq 0.22$), but both recognition memories were intact over time ($t \geq 3.16$, $p \leq 0.03$). In the hemiparkinsonian rats with PD cholinopathy, long-lasting impairment of spatial memory ($X^2 \geq 0.72$, $p \geq 0.22$) was followed by delayed short- and long-term impairment of recognition memory ($t=-0.24$, $p=0.82$) along with hippocampal PV suppression ($z=-3.17$, $p=10^{-3}$), which was functionally coupled to impairment of recognition memory ($r=0.52$, $p=0.04$).

Our results suggest that dopaminergic denervation plays an important role in impairing spatial memory in the prodromal stage of PD, whereas cholinergic denervation and hippocampal PV suppression impair short- and long-term memory in a delayed manner in PD cholinopathy and hemiparkinsonism with PD cholinopathy.

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