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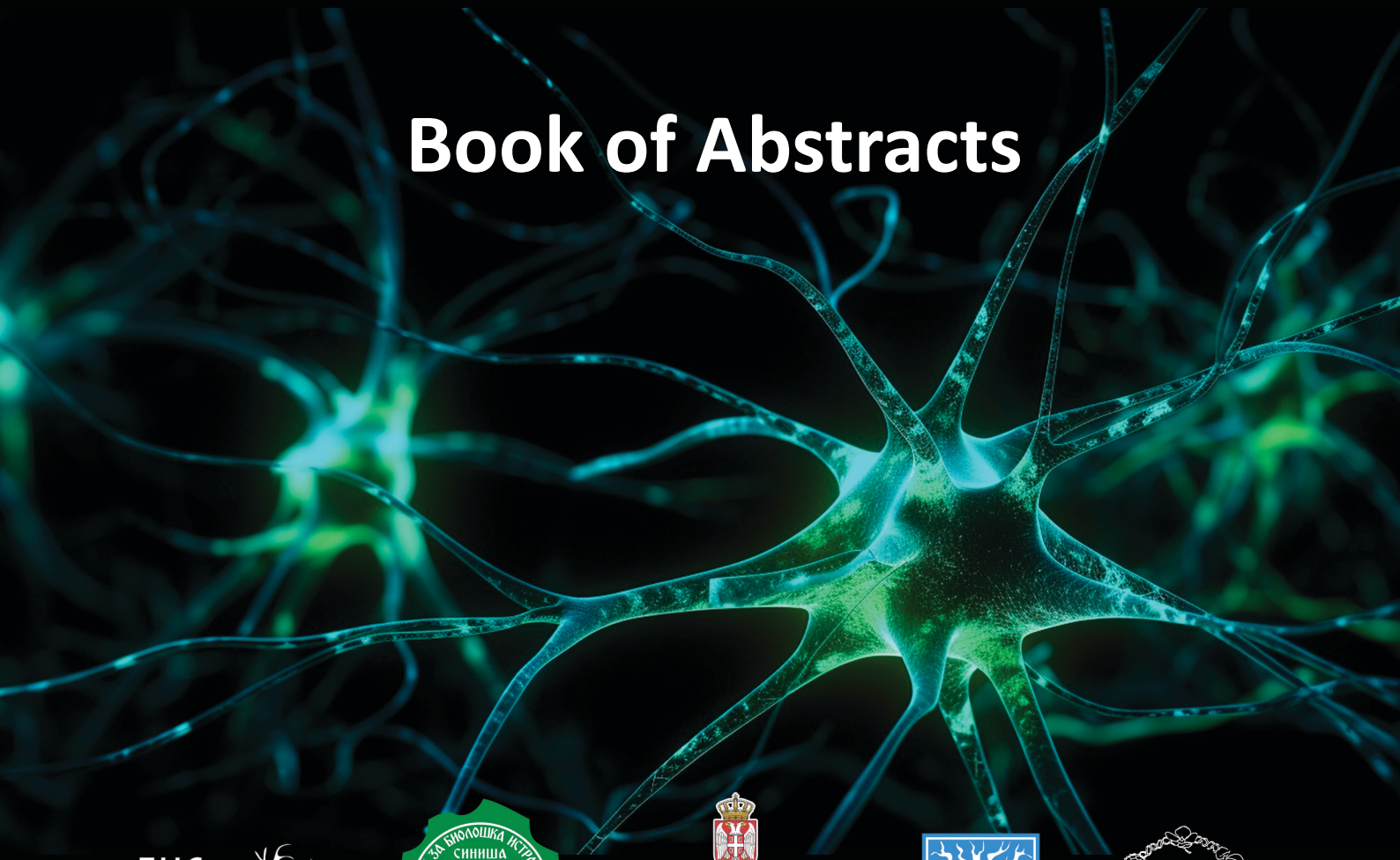

Book of Abstracts

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Distinct clinical outcomes of Complete Freund's adjuvant-free experimental autoimmune encephalomyelitis induced in DA rats

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Experimental autoimmune encephalomyelitis (EAE) is commonly induced with central nervous system antigens mixed with complete Freund's adjuvant (CFA). This adjuvant has a confounding influence on the translational capacity of EAE as multiple sclerosis (MS) model. Thus, we developed a novel subtype of EAE induced in Dark Agouti (DA) rats with spinal cord homogenate (SCH) without CFA and characterized it as a reliable MS model. Despite genetic homogeneity of experimental animals and controlled environmental conditions, we observed variations in EAE clinical course in SCH-immunized DA rats and four clinical groups were identified: lethal, severe, moderate, and mild. Immune cells of spinal cord, small intestine lamina propria and lymph nodes draining the site of immunization were compared between moderate and severe group. Higher numbers of $CD4^+$ T cells, regulatory T cells (Treg), helper T cells type 1 (Th1) and 17 (Th17), and B cells were detected in the spinal cords of severe group. Also, higher levels of interferon (IFN)-γ and interleukin (IL)-6 and an increased proportion of Th1 and Th17 cells were detected in the lamina propria of the severe group. Aminoguanidine – an inducible nitric oxide synthase inhibitor that was applied to the rats during the effector phase of the disease ameliorated EAE and imposed a shift of clinical outcomes towards milder variants. Our results suggest that different clinical outcomes in DA rats come as a consequence of variability in the strength of the effector mechanisms exerted within the CNS. The study of the underlying mechanisms for the observed variability is necessary.

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