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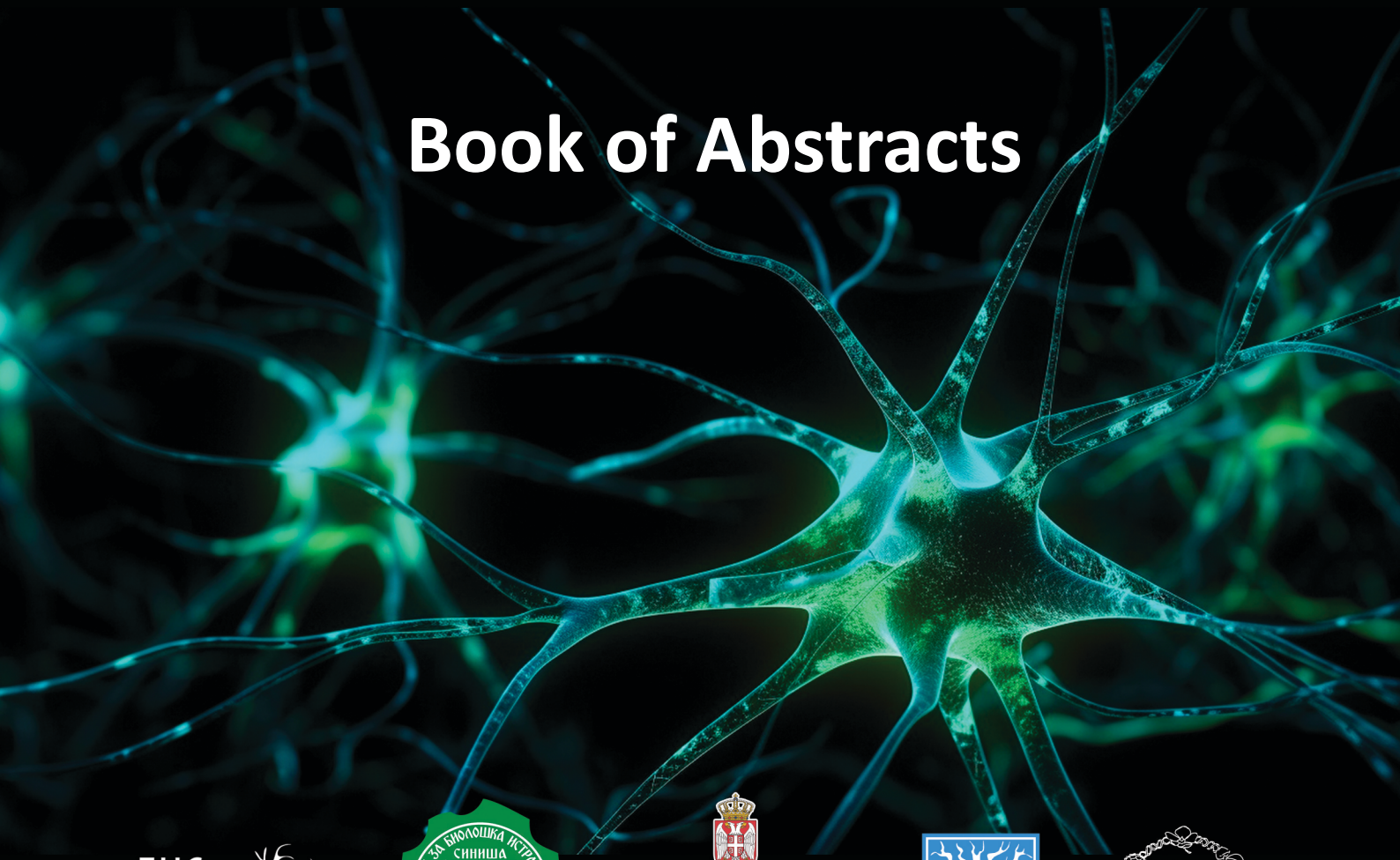

Book of Abstracts

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Effects of cuprizone-induced demyelination on autophagy markers in different neural structures with the evaluation of behavior in rats

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Introduction: Cuprizone is a copper chelating agent rensposible for toxic demyelination. Although its mechanism of demyelination is not fully elucidated, its toxicity is thought to result, at least partially, from induced autophagy.
The Aim: To examine the effect of cuprizone on autophagy markers in different neural structures in rats with the assessment of behavior in the open field test.
Material and Methods: This study was performed on 16 female DA rats, 6 weeks old.The control group (n=8) did not receive cuprizone, while the experimental group (n=8) received food with 0.6% cuprizone content during 7 weeks. Afterwards, locomotion and anxiety-like behavior were assessed in the open field test. Imunoblot technique was performed on: cortex, corpus callosum, cerebellum and spinal cord, in order to examine the markers of autophagy signaling pathway: pUlk, p62, Beclin1, LC3II, pAMPK, pmTOR and pRaptor.
Results: Cuprizone-fed animals had a reduced locomotor activity and exhibited an anxiety-like behavior. In the cortex, p62 and pAMPK were decreased, while pmTOR tended to be lower. In the corpus callosum, pULK was increased and pmTOR was decreased. LC3II tended to be increased, while pAMPK tended to be decreased. In the cerebellum, pAMPK was reduced, while pmTOR was increased. In the spinal cord, Beclin1 and pRaptor were decreased, while pmTOR, pAMPK, LC3II, p62 tended to be decreased.
Conclusion: Long-term use of cuprizone leads to impaired locomotor activity and anxiety-like behavior. Cuprizone-induced damage is most likely due to AMPK-independent autophagy in the cortex and corpus callosum, while autophagy is probably inhibited in the cerebellum and spinal cord.

Keywords: cuprizone; demyelination; autophagy; behavior