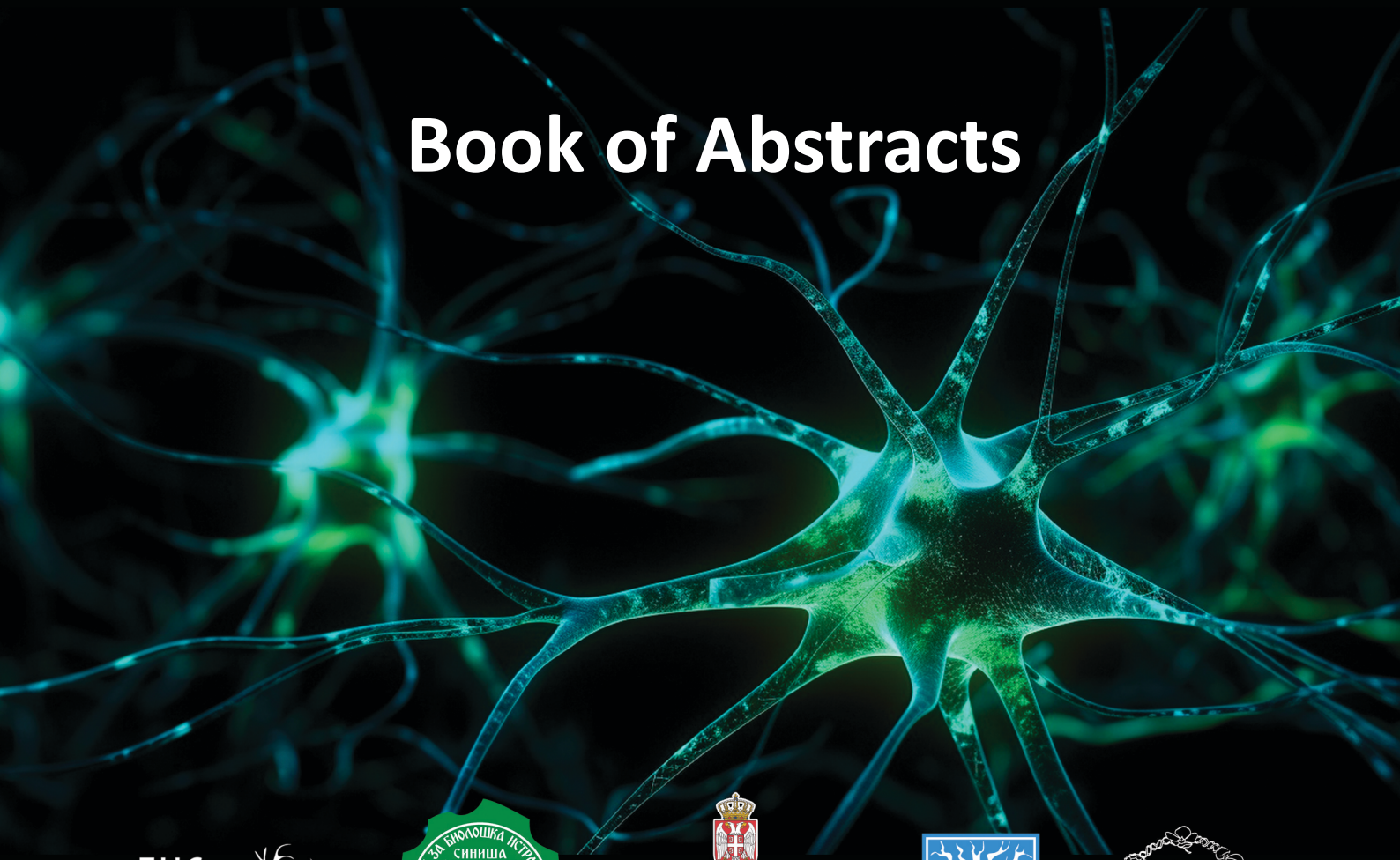
ДНС Друштво за неуронауке Србије
SNS Serbian Neuroscience Society

8

Congress Serbian Neuroscience Society

31 May - 02 June
Belgrade Youth Center
Belgrade



Book of Abstracts

ДНС SNS

Институт за биолошка истраживања Синиша Станковић ИБИСС Универзитет у Београду

Република Србија Министарство науке, технолошког развоја и иновација

Универзитет у Београду Медицински факултет University of Belgrade Faculty of Medicine

Labena

PROMEDIA Laboratory supply specialists

ZEISS

8th CONGRESS OF SERBIAN NEUROSCIENCE SOCIETY with international participation

31 May – 2 June 2023. Belgrade, Serbia - BOOK OF ABSTRACTS

Published by:
Serbian Neuroscience Society
Bulevar despota Stefana 142, 11060 Belgrade, Serbia

Editors
Selma Kanazir and Danijela Savić

Assistant editors:
Anica Živković
Željko Pavković

Technical editor:
Anđela Vukojević

Graphic design:
Olga Dubljević, Irina Veselinović

ISBN: 978-86-917255-4-9

Ecto-5'-nucleotidase marks amoeboid microglial cells in the rat model of neurodegeneration

Ivana Grković[1], Milorad Dragić[2], Nataša Mitrović[1], Marina Zarić Kontić[1], Jelena Martinović[1], Ivana Guševac Stojanović[1]

[1]*Department of molecular biology and endocrinology, VINČA Institute of Nuclear Sciences - National Institute of the Republic of Serbia, University of Belgrade, Belgrade, Serbia*
[2]*Department for General Physiology and Biophysics, Faculty of Biology, University of Belgrade, Belgrade, Serbia.*

Adenosine 5'-triphosphate (ATP) and adenosine are versatile signaling molecules involved in many pathophysiological processes in the nervous system. They can be released from all types of brain cells in the extracellular space and activates purinergic receptors. Signaling via extracellular ATP is regulated by cell-surface located ectonucleotidases. Extracellular AMP resulting from the hydrolysis of ATP and ADP can in turn be hydrolyzed into adenosine by ecto-5'-nucleotidase (eN). We examined the involvement of purinergic signaling components in the rat model of trimethyltin (TMT)-induced hippocampal neurodegeneration (8mg/kg, single ip), which results in behavioral and neurological dysfunction similar as in Alzheimer's disease models. Enzyme histochemistry and immunohistochemistry (ir) showed that products of AMPase activity and eN-ir were accumulated in the neuronal strata, infiltrating within neuronal cell layers, depicting individual round-shaped elements that covered neuronal layers with pronounced cell death mostly at the late stage of TMT-induced neurodegeneration. Co-localization with $Iba1^+$ specifically marked eN at amoeboid microglial cells. Neither of the tested pro-inflammatory cytokines (IL-1β, TNF-α, IL-10) and C3 nor polarization marker iNOS was found in association with those Iba1/eN^+-cells. Iba1-ir cells co-localized with Arg1-ir and phagocytic marker CD68-*ir*. Marked induction of $P2Y_{12}R$-, $P2Y_6R$-, and $P2X_4$-mRNA at the early stage of TMT-induced neurodegeneration might reflect the migration, and chemotaxis of microglia, while induction of $P2X_7R$ at amoeboid cells probably modulates their phagocytic role. These findings may contribute to a better understanding of the involvement of purinergic signaling components in the progression of neurodegenerative disorders that could be target molecules for development of novel therapies.

Acknowledgement: This work was funded by the Ministry of Science, Technological Development and Innovation of the Republic of Serbia, contracts: 451-03-47/2023-01/200017.