ДНС Друштво за неуронауке Србије
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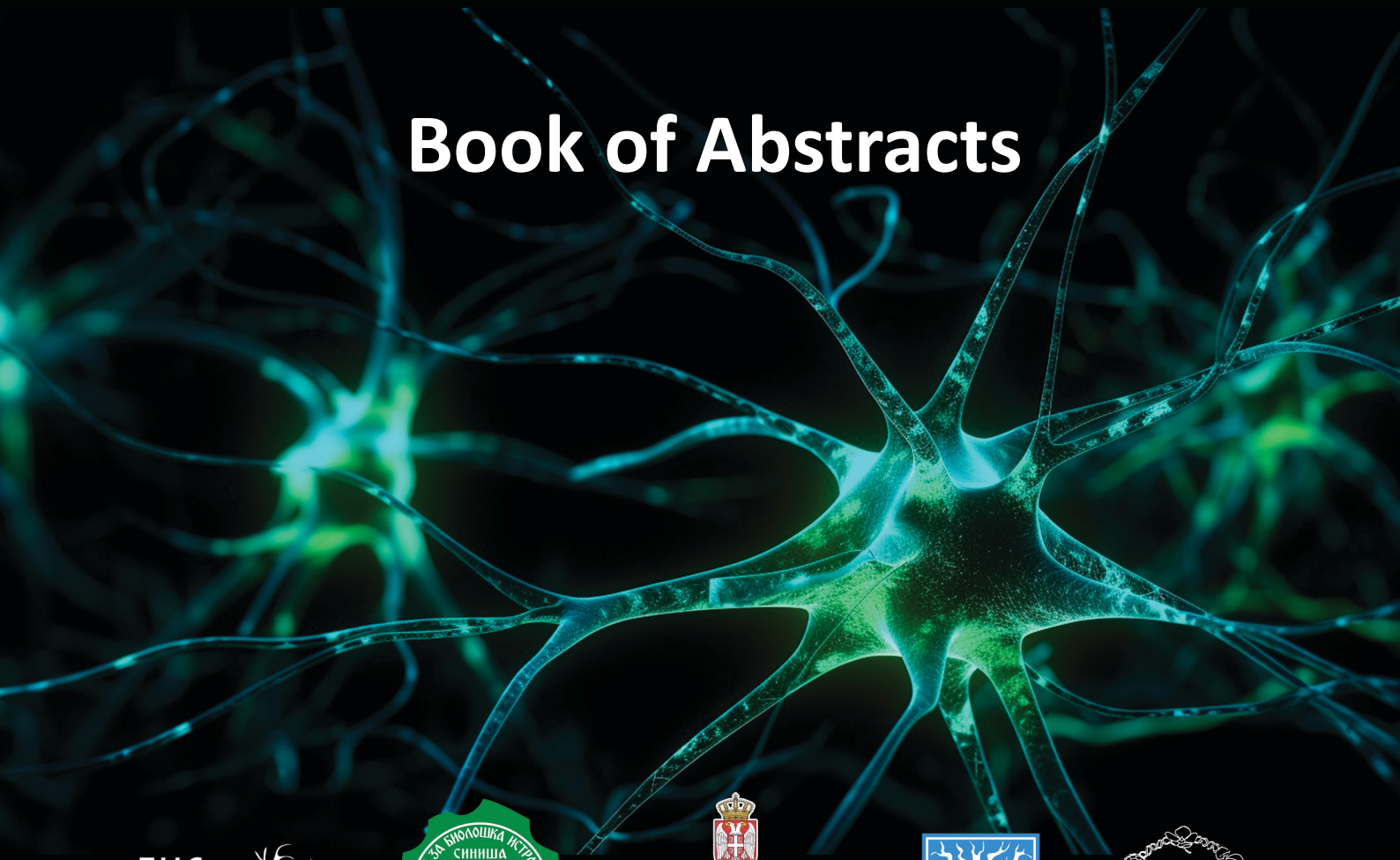

Book of Abstracts

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

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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

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$\alpha_V\beta_3$-Integrin and mitochondria mediate astrocyte response to autoreactive immune cells

Katarina D. Milicevic[1,*], Danijela B. Bataveljic[1], Jelena J. Bogdanovic Pristov[2], Pavle R. Andjus[1], Ljiljana M. Nikolic[3]

[1]*Center for Laser Microscopy, Institute for Physiology and Biochemistry "Jean Giaja", Faculty of Biology, University of Belgrade, Belgrade, Serbia*
[2]*Department of Life Sciencies, Institute for Multidisciplinary Research, University Belgrade, Belgrade, Serbia*
[3]*Department of Neurophysiology, Institute for Biological Research Sinisa Stanovic, National Institute of Republic of Serbia, University of Belgrade, Belgrade, Serbia*
k.milicevic@bio.bg.ac.rs

The astrocytic network maintains homeostasis in the central nervous system (CNS) through interactions with neighboring cells. In the CNS autoimmune disease, multiple sclerosis (MS), neuroinflammatory conditions modulate these cell-to-cell interactions. Our previous work revealed that the immune cells infiltrated into the CNS (CNS-IICs) of experimental autoimmune encemphalomyelitis (EAE) rat, an animal model of MS, rapidly alter the activity pattern of astrocytes by activating the glial P2X7 receptor (P2X7R). In the present study we further defined the mechanisms responsible for astrocytes' activation in the presence of CNS-IICs. For this purpose, we used an *in vitro* experimental setup and monitored Ca^{2+} dynamics in Fluo-4-labeled cultured naïve astrocytes following brief bath application of CNS-IICs isolated from the spinal cord of the EAE rat. Our data indicate that the astroglial $\alpha_v\beta_3$-integrin is involved in the initial contact of astrocytes with CNS-IICs, since blocking $\alpha_v\beta_3$-integrin reduced the expected astrocytic Ca^{2+} response. Furthermore, blocking of mitochondrial Na^+/Ca^{2+}- and H^+/Ca^{2+}- exchangers in astrocytes promoted an augmentation of the intracellular Ca^{2+} increase and a higher ATP release after brief exposure to CNS-IICs, demonstrating that mitochondria regulate the astrocyte-CNS IICs cell-cell interaction. Overall, our study expands the understanding of astrocytes' interaction with autoreactive immune cells that are present in their local environment in an autoimmune disease. This offers a new conceptual framework for considering direct astrocyte–immune cell interaction to design new strategies for therapy development in the treatment of MS.

Acknowledgements: This work was supported by the Ministry of Science, Technological Development and Innovation of Republic of Serbia (Contract no. 451-03-47/2023-01/200007, 451-03-47/2023- 01/200053 and 451-03-47/2023-01/200178).