ДНС Друштво за неуронауке Србије
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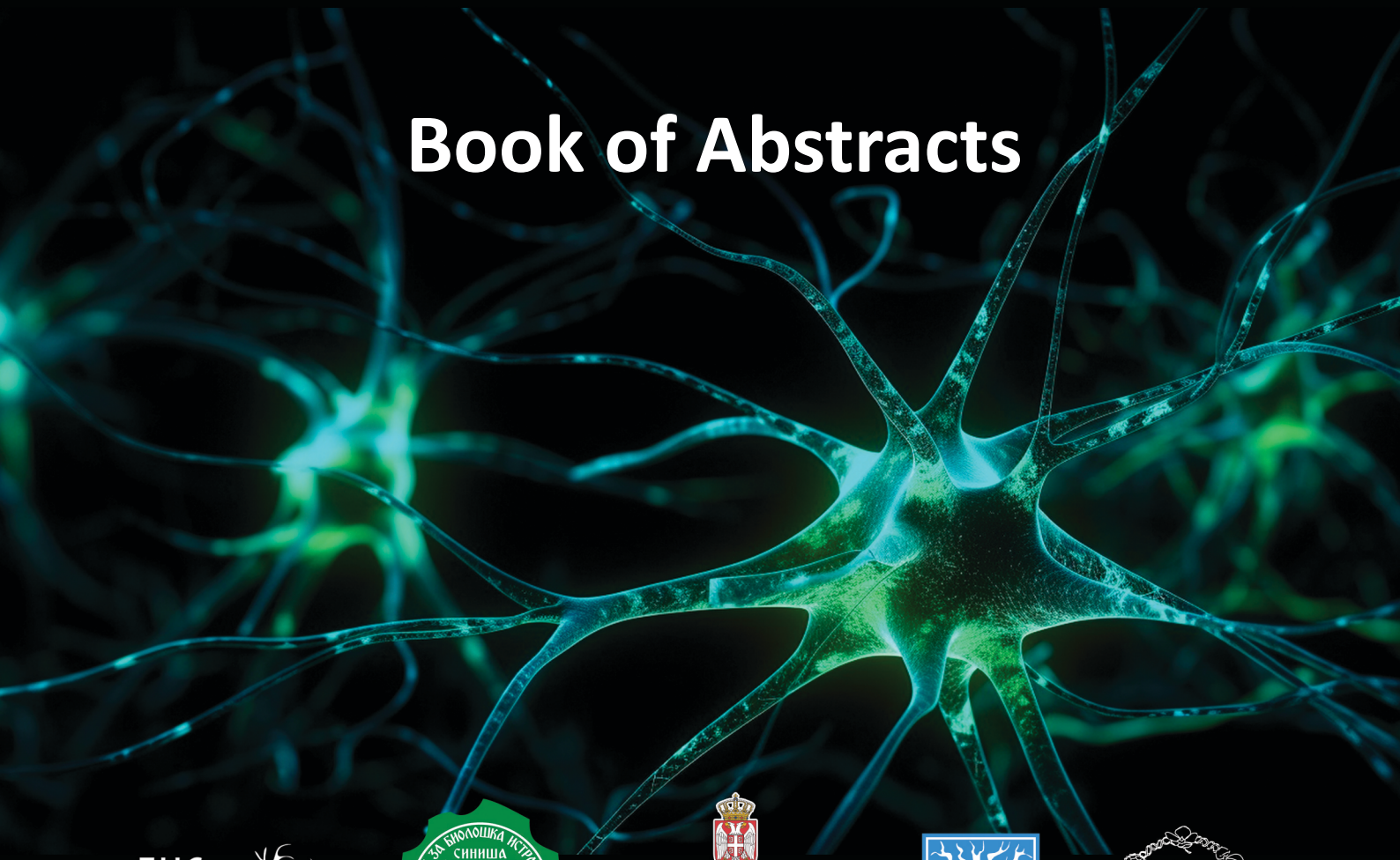

Book of Abstracts

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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Thyroid hormone metabolism in the cortex of male and female APP knock-in mice

Jelena Ciric[1,*], Nikola Milovanovic[1], Irena Jovanovic Macura[1], Vesna Tesic[3], Branko Filipovic[2], Branka Sosic-Jurjevic[2], Milka Perovic[1]

[1] *Laboratory for Molecular Neurobiology and Behavior, Department of Neurobiology, Institute for Biological Research "Siniša Stanković", National Institute of the Republic of Serbia, University of Belgrade, Belgrade, Serbia;*
[2] *Department of Citology, Institute for Biological Research "Siniša Stanković", National Institute of the Republic of Serbia, University of Belgrade, Belgrade, Serbia*
[3] *Department of Neurology, LSU Health Sciences Center - Shreveport, Biomedical Research Institute (BRI), Shreveport, USA*
jelena.ciric@ibiss.bg.ac.rs

Targeting novel pathways contributing to the pathogenesis/progression of Alzheimer's disease (AD) is crucial due to the lack of effective management and treatment modalities. Higher prevalence, progression rate and severity of AD in women than in men also establish sex as key variable in AD therapy development.

Thyroid disorders, both hyper- and hypothyroidism, were found to occur with up to nine-fold higher prevalence in women compared to men. The molecular mechanisms by which thyroid dysfunction contribute to AD pathogenesis and heterogeneity remain however elusive. We therefore examined sex-related alterations in gene expression of iodothyronine deiodinase 2 (Dio2) and transthyretin (TTR) involved in the tissue metabolism and the distribution of thyroid hormones (THs), respectively, in a novel, state-of the art knock-in (KI) mouse model of AD-like amyloidosis, $APP^{NL\text{-}G\text{-}F}$ mice.

Quantitative RT-PCR analysis revealed prominent differences in cortical Dio2 and TTR gene expression in 9-month-old male and female $APP^{NL\text{-}G\text{-}F}$ mice and their non-KI littermates (WT). In comparison to WT male mice, the increase in *Dio2* mRNA level was evident in female WT mice, while a trend toward a decrease was detected in their $APP^{NL\text{-}G\text{-}F}$ KI littermates. Expression in the opposite direction was observed for TTR, with a robust genotype-dependent decrease in male mice.

Results are in line with well-established role of THs in the regulation of neuronal plasticity in the adult brain and suggest profound sex-biased effects of TH on Aβ-induced pathology in $APP^{NL\text{-}G\text{-}F}$ mice.

Acknowledgement: This study was financed by the Ministry of Science, Technological Development and Innovation of the Republic of Serbia: 451-03-47/2023-01/200007.