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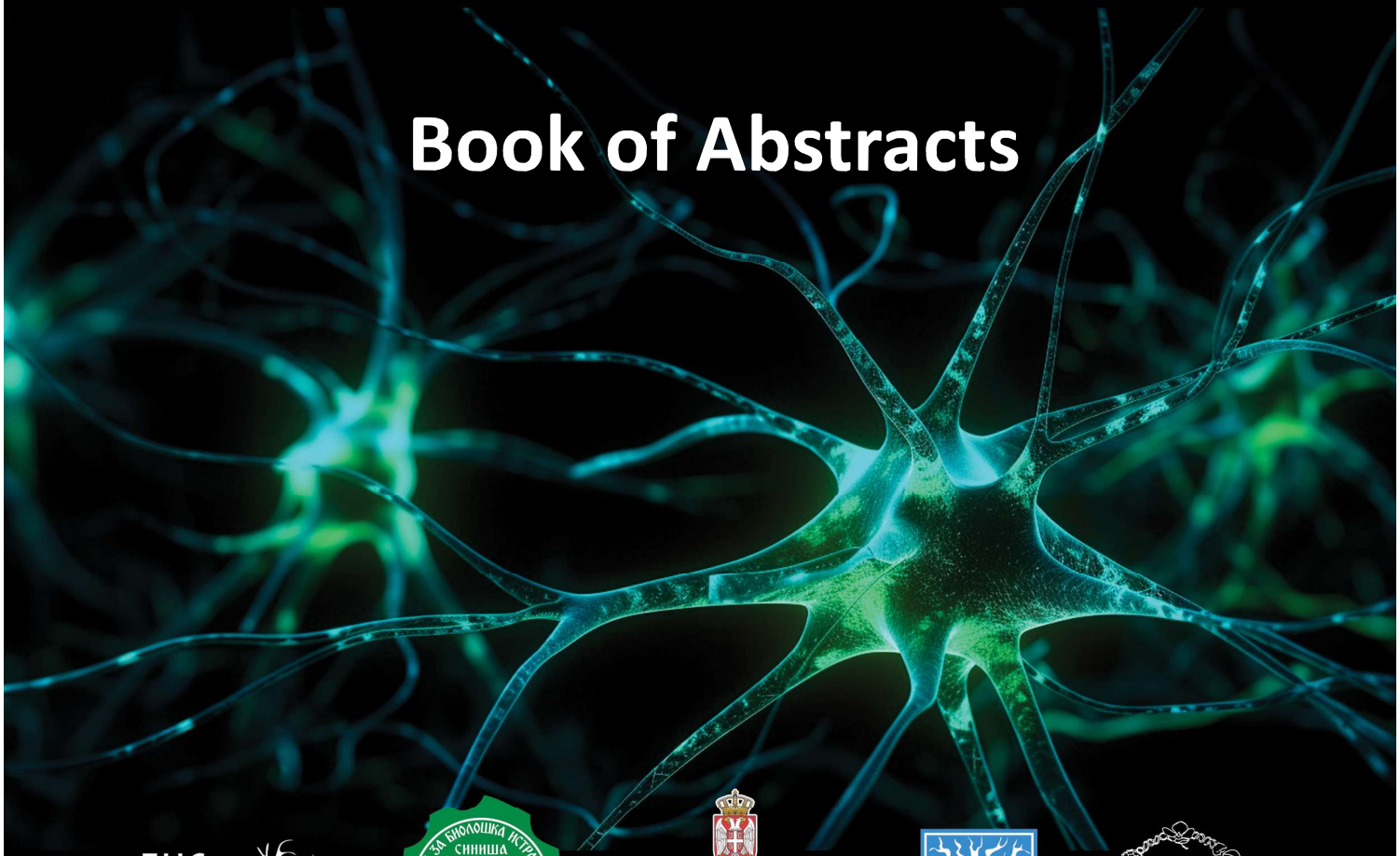

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Agmatine upregulates Nrf2/HO-1 pathway in Lps-stimulated microglia

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Oxidative burst is a component of neuroinflammation whereby microglia-generated reactive oxygen species (ROS) either target pathogens or act as secondary messengers for microglial activation. In response to increased ROS during microglial activation, cytoprotective mechanisms are initiated primarily via Nrf2 activation and HO-1 expression. Agmatine is known to exert neuroprotective effect in vivo due to Nrf2 induction. While agmatine has been shown to activate the Nrf2/HO-1 signaling and protect macrophages from Lps-induced inflammation in vitro, its interaction with this pathway in activated microglia remains unexplored.

Therefore, we sought to examine the potential of 100 μM agmatine as a pretreatment of Lps to activate Nrf2 in BV-2 microglia. In addition to cell viability, we analyzed the nuclear level of Nrf2 by Western blot and the expression of *Hmox1* by PCR, as well as the protein level of HO-1. We also measured indicators of prooxidant and antioxidant activity: 4-HNE and total glutathione, respectively.

Agmatine induces oxidative stress in non-stimulated microglia, as confirmed by the increase in the lipid peroxidation marker — 4-HNE, while cell viability stays preserved. Moreover, agmatine alone causes delayed Nrf2 nuclear overexpression and an increase in total glutathione content, eventually leading to an adaptive stress response. On the other hand, in Lps-stimulated microglia, agmatine prevents lipid peroxidation, readily upregulates the nuclear protein levels of Nrf2, which increases gene and protein expression of HO-1, and maintains delayed Nrf2 nuclear overexpression, resulting in increased total glutathione content associated with cytoprotection.

Overall, we interpret agmatine-induced oxidative stress in non-activated microglia as triggering the adaptive response via Nrf2 and total glutathione, enabling them to cope with subsequent stressors, ie, Lps.

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