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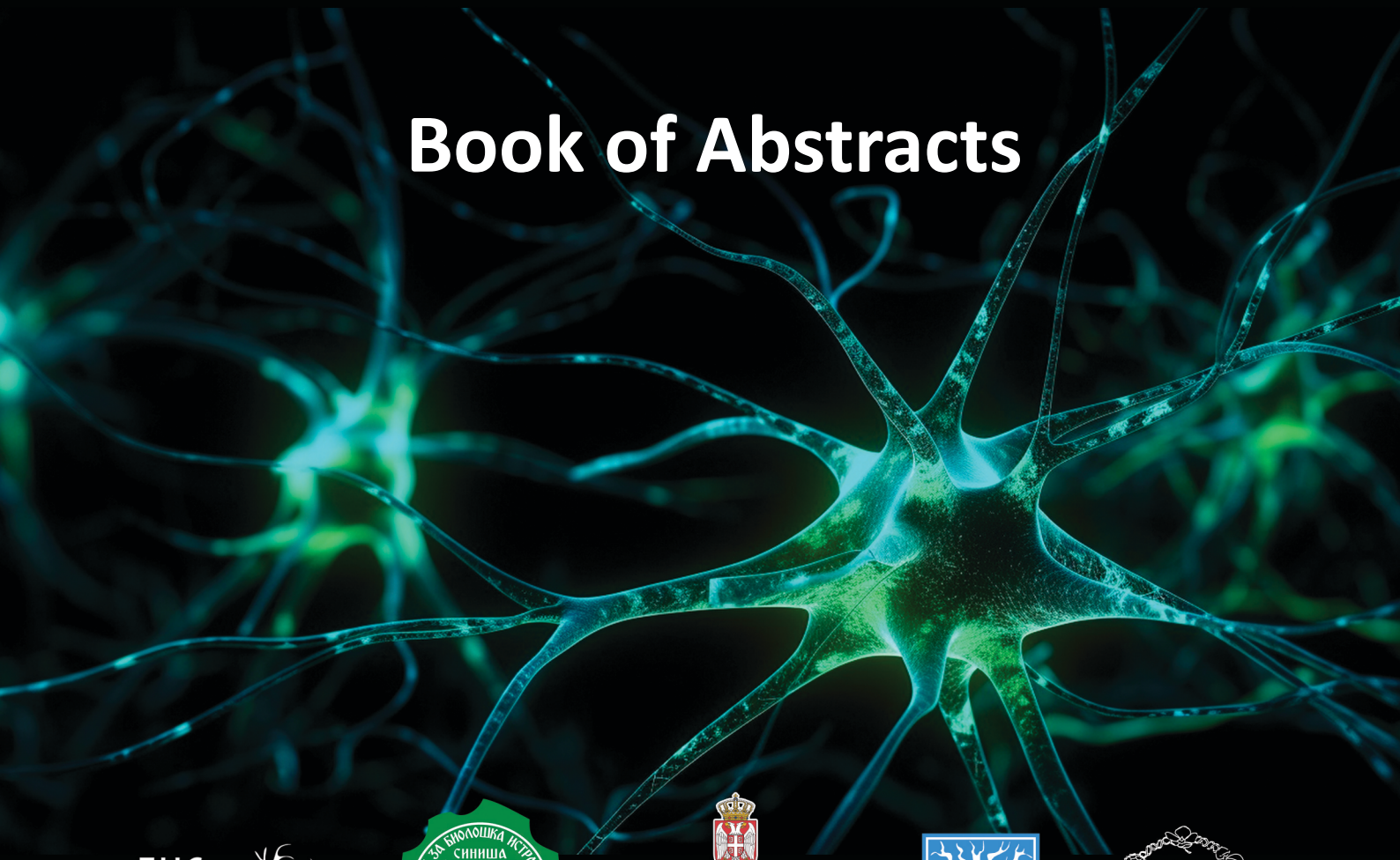

# Book of Abstracts

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# Short-term fish oil treatment increases number of microglial cells and expression level of TREM-2 in parietal cortex of 5XFAD mice

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According to the amyloid hypothesis of Alcheimer's disease (AD), disruption of balance between production and clearance of Aβ from the cells leads to the progressive accumulation and aggregation of amyloid beta (Aβ) peptides in the brain. The glial system (microglial cells and astrocytes) is responsible for maintaining homeostasis in the brain which implies its important role in the development and progression of AD. Our previous work revealed that the short-term fish oil (FO) treatment in 5xFAD mice, (AD animal model), reduces toxic Aβ load and increases number of microglial/macrophage cells in parietal cortex. In the present study we aimed to further decipher the roles of microglial and macrophage cells and to elucidate possible mechanisms responsible for observed reduced level of toxic Aβ42 peptide. For this purpose, western blot and immunohistochemical analysis were used to detect changes in parietal cortex of three-month-old 5xFAD mice after three weeks FO treatment (100µl/animal/day). Distinction between microglial cells and macrophages was assessed using double immunostaining with anti-TMEM119 and anti-Iba1 antibodies respectively. Immunostaining was observed by confocal microscopy. For western blot analysis anti-TREM-2 and anti-IDE were used to observe potential mechanism responsible for extracellular clearance of toxic Aβ forms. Quantification was done by Image Quant software. Our results showed that short-term FO supplementation affects the localization and number of microglial cells and macrophages. Macrophages were located around the plaque and were responsible for the formation of a mechanical barrier, while microglial cells showed an increased number under the treatment and were located far from the plaques. Furthermore, the treatment did not seem to affect the level of IDE, while on the other hand it significantly increased the level of TREM-2 (ultimately sustaining the microglial response to Aβ-plaque-induced pathology).

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