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Microglia activation in animal model of post-traumatic stress disorder

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The central nervous system has historically been considered immune-privileged; however this privileged position mostly consists of adaptive immune responses with restricted access of infiltrating lymphocytes into the brain parenchyma, while cells of the innate immune system; microglia are abundant in the brain. Microglia is also considered a key player in many neuro-inflammatory conditions. Microglial cells respond to infectious agents such as LPS with reactive phenotype and changes in expression of certain markers such as Iba1. Reactive microglia is also found in the brain during neuro-inflammatory processes in depression, bipolar disorder, Post-Traumatic Stress Disorder (PTSD). We used animal model of PTSD in order to test how chronic stress affects the neurogenesis, reactivity of microglial cells as well as their density in the dentate gyrus of the hippocampus and whether hippocampal volume is changing during PTSD. According to our results, 10 days after stress onset the number of Iba1+ microglial cells in the dentate gyrus of the hippocampus increased substantially compared to the control group (Mann-Whitney, p=0,028). However we did not see any inflammatory foci, i.e. microglial nodules. The intensity of Iba1+staining of as well as the size and shape of cells did not differ from the control group. The hippocampal volume did not change significantly. We propose that neurotoxic or neuro-protective role of microglia cells can change depending on the microenvironment, such as in presence of certain cytokines, interleukins, hormones that lead to corresponding changes in the molecular profiles of glial cells. The particular mechanisms of microglia activation and its role in neurogenesis are discussed.

Biography

Oksana Tuchina is a senior post-doctorate neurobiologist at Immanuel Kant Baltic Federal University (IKBFU). Her research interest focuses on neurobiology and human physiology. She has more than 12 publications to her credit.

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