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## Poster Presentation

### Microglia in Traumatic Brain Injury

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#### Abstract

Microglia is one of the first innate immune components. These cells account about 5 to 10% of the entire adult brain cells and are activated by trauma. Complex-mediated inflammatory responses occur through cellular and molecular events during and after the traumatic brain injury (TBI). In-lesion area astrocytes, microglia, and damaged neurons begin to secrete cytokines and chemokines. Microglia has the potential to polarize the M1-like and M2-like phenotypes. Several studies have been shown that the use of different therapeutic methods effect on the polarization of microglia phenotypes. Intracranial transplantation of human neural stem cells (hNSCs) decreased microglial activity through M2/M1 ratio in the cortical-controlled injury model. This switching of phenotype was associated with an increase in the expression of the anti-inflammatory interleukin-4 receptor  $\alpha$  and a decrease in the expression of the proinflammatory interferon- $\gamma$  receptor  $\beta$ , and ultimately most hNSCs differentiated into neurons. Microglia has proposed as a target cell in the process of treatment after head trauma. Different phenotypes of microglia have different effects on the tissue and brain function. Knowing how microglia works on neurodegenerative and TBI diseases are crucial for determining therapeutic strategies.

**Keywords:** Microglia, Traumatic Brain Injury, Cell Therapy

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