



# The 1<sup>st</sup> International Neuroinflammation Congress and 1<sup>st</sup> Student Festival of Neuroscience

Shefa Neuroscience Research Center, Tehran, Iran, 11-13 April, 2017

*The Neuroscience Journal of Shefaye Khatam*

Volume 5, No. 2, Suppl 2

## Poster Presentation

### Assay of Alterations of Cytokines to Remedy of Traumatic Brain Injury

Fateme Salahshour\*, Shima Ghari

Islamic Azad University, Mashhad Branch, Mashhad, Iran

**Published: 11 April, 2017**

#### **Abstract**

Traumatic brain injury (TBI) is a global health concern that typically causes emotional disturbances and cognitive dysfunction. It elicits a complex secondary injury response, with neuroinflammation as a crucial central component. Secondary pathologies following TBI may be associated with chronic neurodegenerative disorders and an enhanced likelihood of developing dementia-like disease in later life. The damage to the brain occurs in two phases, the initial primary phase being the injury itself, which is irreversible and amenable only to preventive measures to minimize the extent of damage, followed by an ongoing secondary phase, which begins at the time of injury and continues in the ensuing days to weeks. This delayed phase leads to a variety of physiological, cellular, and molecular responses aimed at restoring the homeostasis of the damaged tissue, which, if not controlled, will lead to secondary insults. Many of the issues that TBI patients face are thought to be mediated by the immune system. TBI induced a moderate increase in both pro- and anti-inflammatory cytokines/chemokines. Estrogen therapy following diffuse TBI has led to reducing pro-inflammatory cytokines while induced the brain IL-10 level, and the changes of cytokines by estrogen may be regarded as one of the action mechanisms of its antiedema effect. Post-injury administration of MW151 that induced overproduction of proinflammatory cytokines towards homeostasis without immunosuppression in a closed head injury model of mild TBI suppressed acute cytokine up-regulation and downstream cognitive impairment. The development of novel treatments following TBI should aim at minimizing secondary injury by modifying, rather than eliminating the inflammatory response, while creating optimal conditions for regeneration to date there is no effective treatment available to patients, and morbidity and mortality remain high.

**Keywords:** TBI, Neuroinflammation, Cytokines, Treatment

**\*Corresponding Author:** Fateme Salahshour

**E-mail:** salahshourfateme@gmail.com