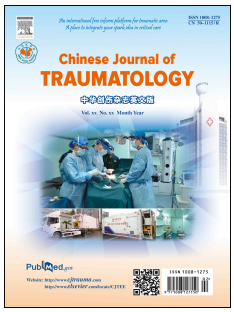
# Accepted Manuscript




Current understanding of neuroinflammation after traumatic brain injury and cell-based therapeutic opportunities

Ye Xiong, Asim Mahmood, Michael Chopp

PII: S1008-1275(18)30058-0

DOI: 10.1016/j.cjtee.2018.02.003

Reference: CJTEE 314

To appear in: *Chinese Journal of Traumatology*

Received Date: 13 February 2018

Revised Date: 2 March 2018

Accepted Date: 5 March 2018

Please cite this article as: Xiong Y, Mahmood A, Chopp M, Current understanding of neuroinflammation after traumatic brain injury and cell-based therapeutic opportunities, *Chinese Journal of Traumatology* (2018), doi: 10.1016/j.cjtee.2018.02.003.

**Review article**

Article history
Received 13 February 13 2018
Received in revised form 2 March 2018
Accepted 5 March 2018

# Current understanding of neuroinflammation after traumatic brain injury and cell-based therapeutic opportunities

Ye Xiong[a]*, Asim Mahmood[a], Michael Chopp[b,c]

a. Department of Neurosurgery Henry Ford Health System, 2799 West Grand Boulevard, Detroit, MI 48202, USA
b. Department of Neurology, Henry Ford Health System, 2799 West Grand Boulevard, Detroit, MI 48202, USA.
c. Department of Physics, Oakland University, Rochester, MI 48309, USA

*Corresponding author: Email: yxiong1@hfhs.org

**Abstract**

Traumatic brain injury (TBI) remains a major cause of death and disability worldwide. Increasing evidence indicates that TBI is an important risk factor for neurodegenerative diseases including Alzheimer's disease, Parkinson's disease, and chronic traumatic encephalopathy. Despite improved supportive and rehabilitative care of TBI patients, unfortunately, all late phase clinical trials in TBI have yet to yield a safe and effective neuroprotective treatment. The disappointing clinical trials may be attributed to variability in treatment approaches and heterogeneity of the population of TBI patients as well as a race against time to prevent or reduce inexorable cell death. TBI is not just an acute event but a chronic disease. Among many mechanisms involved in secondary injury after TBI, emerging preclinical studies indicate that posttraumatic prolonged and progressive neuroinflammation is associated neurodegeneration which may be treatable long after the initiating brain injury. This review provides an overview of recent understanding of neuroinflammation in TBI and preclinical cell-based therapies that target neuroinflammation and promote functional recovery after TBI.