The 1st International Neuroinflammation Congress and 1st Student Festival of Neurosience

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

The Neuroscience Journal of Shefaye Khatam

Volume 5, No. 2, Suppl 2

Poster Presentation

Apoptosis Following Cortical Spreading Depression in Juvenile Rats

Ali Jahanbazi Jahan-Abad[1*], Leila Alizadeh[1], Sajad Sahab Negah[1,2], Parastoo Barati[1], Maryam Khaleghi Ghadiri[3], Sven G Meuth[4], Stjepana Kovac[4], Ali Gorji[1,2,3]

[1]Shefa Neuroscience Research Center, Khatam Alanbia Hospital, Tehran, Iran
[2]Department of Neuroscience, Mashhad University of Medical Sciences, Mashhad, Iran
[3]Department of Neurosurgery, Wilhelms-Universität Münster, Münster, Germany
[4]Department of Neurology, Westfälische Wilhelms-Universität Münster, Münster, Germany
[5]Epilepsy Research Center, Westfälische Wilhelms-Universität Münster, Münster, Germany

Published: ***11 April, 2017***

Abstract

Introduction: Repetitive cortical spreading depression (CSD) can lead to cell death in immature brain tissue. Caspases are involved in neuronal cell death in several CSD-related neurological disorders. Yet, whether repetitive CSD itself can induce caspase activation in adult or juvenile tissue remains unknown. Inducing repetitive CSD in somatosensory cortices of juvenile and adult rats *in vivo*, we thus aimed to investigate the effect of repetitive CSD on the expression of caspases 3, 8, 9, and 12 in different brain regions. **Materials and Methods:** Toluidine Blue staining and TUNEL assay were done to measure the mean number of dark neurons formation and the mean number of TUNEL positive cells in CSD rat models. Neuronal apoptosis assay was performed by immunohistochemistry and western blotting assay. **Results:** Higher numbers of dark neurons and TUNEL-positive cells were observed in the hippocampal CA1 and CA3 regions as well as in the entorhinal and somatosensory cortices after CSD in juvenile rats. This was accompanied by higher expression of caspases 3, 8, and 9. Caspase-12 levels remained unchanged after CSD, suggesting that endoplasmic reticulum stress is not involved in CSD-triggered apoptosis. Changes in caspase expression were paralleled by a decrease of procaspase-3, -8 and -9 in juvenile rat brain tissue subjected to CSD. In contrast, repetitive CSD in adult rats did not result in the upregulation of caspase signaling. **Conclusion:** Our data points to a maturation-dependent vulnerability of brain tissue to repetitive CSD with a higher degree of apoptotic damage and caspase upregulation observed in juvenile tissue. Findings suggest a key role of caspase signaling in CSD-induced cell death in the immature brain. This implies that anti-apoptotic treatment may prevent CSD-related functional deficits in the immature brain.

Keywords: Cortical spreading depression, Cell death, Caspases, Dark neurons, Apoptosis

Corresponding Author: Ali Jahanbazi Jahan-Abad

E-mail: *a.jahanbazi65@yahoo.com*

[Downloaded from shefayekhatam.ir on 2026-11-28]