The Neuroprotective Roles of NMDA Receptors Antagonist Against with Lesion in Central Nucleus of Amygdala

Milad Ahmadi1,2*, Azadeh Sajadian1, Hadi Aligholi1,2, Babak Khodaie1, Ahmad Ali Lotfinia1, Mahmoud Lotfinia1,4

1Shefa Neuroscience Research Center, Khatam Alanbia Hospital, Tehran, Iran.
2Faculty of Veterinary Medicine, Islamic Azad University, Karaj Branch, Karaj, Iran.
3Department of Neurosciences, School of Advanced Technologies in Medicine, Tehran University of Medical Sciences, Tehran, Iran.
4Shahid Beheshti University of Medical Sciences, Tehran, Iran.

Published: 18 February, 2015

Abstract
The N-Methyl-D-aspartic acid (NMDA) receptor is one of the specific types of ionotropic receptors which lied to glutaminergic system. It has been widely accepted that NMDA receptor neurons promote anxiety, in humans as well as in animal models. However, in the previous study seems inhibiting of this receptors decreased the protective role in neural cell. Demonstration of NMDA receptor activity had neurodegeneration effect. The hypothesis of this study based on evaluated neuroprotectivity effect of NMDA receptor inhibition in the central nucleus of the amygdala (CeA) which have role in anxiety. In this study, we confirm that NMDA antagonist (MK801) is neuroprotective when CeA had confronted regional electrical lesion. Kindling of amygdala by electrical lesion which presented by stimulation of the CeA area after microinjection of MK801 (0.5 and 0.75 µg/kg) in the kindling area is may indicate neuroprotection from secondary injury. It seems the rats were confronted electrical lesion indicated specific decreases in the percentage of open arm time and percentage of open arm entries in elevated plus maze. However, in histopathological study, the mean of dark neurons of CeA in the MK801 group was less than lesion group. We concluded that inhibited of NMDA receptors in CeA neurons may contribute to induced neuroprotection by electrical lesion.

Keywords: NMDA receptor, Neurodegenerative, Dark Neuron.

*Corresponding Author: Milad Ahmadi
E-mail: pmiladz@gmail.com