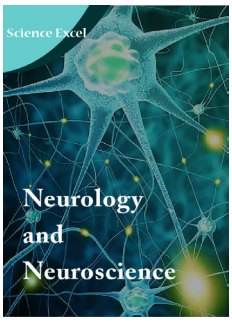
Neurology & Neuroscience

Short Notes



Dexamethasone ameliorates the damage of hippocampal filamentous actin cytoskeleton but is not sufficient to cease epileptogenesis in pilocarpine induced epileptic mice

Yan-Feng Zhang* and Nuo Yang

Department of Pediatric Neurology, the First Hospital of Jilin University, Changchun, Jilin Province 130021, PR China

Correspondence

Yan-Feng Zhang

Department of Pediatric Neurology, the First Hospital of Jilin University, Changchun, Jilin Province 130021, PR China

Tel: 86 15804392859

E-mail: yfzhang@jlu.edu.cn

- Received Date: 23 Feb 2020;
- Accepted Date: 10 Mar 2020;
- Publication Date: 16 Mar 2020.

Progressive deconstruction of filament actin (F-actin) in hippocampal neurons in the epileptic brain have been associated with epileptogenesis. Previous clinical studies suggest that glucocorticoids treatment plays beneficial roles in refractory epilepsy. Glucocorticoids treatment affects dendritic spine morphology by regulating local glucocorticoid receptors and F-actin cytoskeleton dynamics.

In the present study, effects of dexamethasone on actin filament organization are examined in a pilocarpine-induced epileptic model. After treatment with dexamethasone F-actin damage was reduced dramatically in pilocarpine epileptic mice models. Treatment of dexamethasone is beneficial for reducing neuronal loss and maintaining synaptic structures. Dexamethasone treatment improves but is not sufficient to cease epileptogenesis..

Citation: Zhang Y-F, Yang N. Dexamethasone ameliorates the damage of hippocampal filamentous actin cytoskeleton but is not sufficient to cease epileptogenesis in pilocarpine induced epileptic mice. Neurol Neurosci. 2020;1(1):1-1.