Roberto Di Maio, J Neurol Neurophysiol 2015, 6:3
http://dx.doi.org/10.4172/2155-9562.S1.025

OMICS International
conferenceseries.com

4th International Conference and Exhibition on
Neurology & Therapeutics
July 27-29, 2015 Rome, Italy

Post-status epilepticus treatment with the cannabinoid agonist WIN55,212-2 prevents chronic epileptic hippocampal damage in rats

Roberto Di Maio
Purdue University, USA

Repeated seizures are often associated with development of refractory chronic epilepsy, the most common formof which is temporal lobe epilepsy. G-protein-coupled cannabinoid receptors (CB1 and CB2 receptors) regulate neuronal excitability and have been shown to mediate acute anticonvulsant effects of cannabinoids in animal models. However, the potential of cannabinoids to prevent chronic neuronal damage and development of epilepsy remains unexplored. We hypothesized that treatment with a CB receptor agonist after an episode of status epilepticus – but before development of spontaneous recurrent seizures – might prevent the developmentof functional changes that lead to chronic epilepsy. Using the rat pilocarpine model, a therapeutic approach was simulated by administering the CB agonist, WIN55,212-2 after an episode of status epilepticus. Epileptic behavior was monitored during development of spontaneous recurrent seizures for up to 6 months. Histology, neurochemistry, redox status and NMDA receptor subunit expression were assessed at 6 months after pilocarpine-induced seizures. Sub-acute treatment with WIN 55,212-2 (for 15 days starting 24 h after PILOinjection) dramatically attenuated the severity, duration and frequency of spontaneous recurrent seizures.

Further, in contrast to vehicle-treated animals, hippocampi from WIN 55,212-2-treated animals showed: normal thiol redox state, normal NR2A and NR2B subunit expression, preservation of GABAergic neurons and preventionof abnormal proliferation of GABAergic progenitors. This study shows for the first time that, after a known in citing event, treatment with a compound targeting CB receptors has the potential to prevent the epileptogenic events that result in chronic epileptic damage.

rod16@pitt.edu

Notes: