Ik-Hyun Cho, J Alzheimers Dis Parkinsonism 2015, 5:2
http://dx.doi.org/10.4172/2161-0460.S1.013

OMICS International
conferenceseries.com

International Conference on
Parkinson's Disease & Movement Disorders
August 11-13, 2015 Frankfurt, Germany

Sulforaphane ameliorates 3-nitropropionic acid-induced striatal toxicity by activating the Keap1-Nrf2-ARE pathway and inhibiting the MAPKs and NF-κB pathways

Ik-Hyun Cho
Kyung Hee University, Republic of Korea

The potential neuroprotective value of Sulforaphane (SFN) in Huntington's Disease (HD) has not been established. We investigated whether SFN prevents and improves the neurological impairment and striatal cell death in a 3-nitropropionic acid (3-NP)-induced mouse model of HD. SFN (2.5 and 5.0 mg/kg/day, i.p.) was given daily for 30 minutes before 3-NP treatment (pre-treatment) and from onset/progression/peak points of the neurological disorders. Pre-treatment with SFN (5.0 mg/kg/day) produced the best neuroprotective effect for the neurological disorders and lethality among other conditions. The protective effects due to pre-treatment with SFN were associated with suppression of the formation of lesion area, neuronal death, succinate dehydrogenase activity, apoptotic cell death, microglial activation and mRNA or protein expression of inflammatory mediators including tumor necrosis factor-alpha, interleukin (IL)-1β, IL-6, inducible nitric oxide synthase and cyclooxygenase-2 in the striatum after 3-NP treatment. Also, pre-treatment with SFN activated the Kelch-like ECH associated protein 1 (Keap1)-nuclear factor erythroid 2-related factor 2 (Nrf2)-Antioxidant Response Element (ARE) pathway and inhibited the mitogen-activated protein kinases (MAPKs) and nuclear factor-kappa B (NF-κB) pathways in the striatum after 3-NP treatment. As expected, the pre-treatment with activators (dimethyl fumarate and antioxidant response element inducer-3) of the Keap1-Nrf2-ARE pathway decreased the neurological impairment and lethality after 3-NP treatment. Our findings suggest that SFN may effectively attenuate 3-NP induced striatal toxicity by activating the Keap1-Nrf2-ARE pathway and inhibiting the MAPKs and NF-κB pathways and that SFN has a wide therapeutic time-window for HD-like symptoms.

ihcho@khu.ac.kr

Notes: