Nrf2 signaling pathway

It could be postulated that Nrf2-ARE pathway was activated in brain after TBI [1)]

Inflammation is the most common feature of many chronic diseases and complications, while playing critical roles in carcinogenesis. Several studies have demonstrated that Nrf2 contributes to the anti-inflammatory process by orchestrating the recruitment of inflammatory cells and regulating gene expression through the antioxidant response element (ARE). The Keap1 (Kelch-like ECH-associated protein)/Nrf2 (NF-E2 p45-related factor 2)/ARE signaling pathway mainly regulates anti-inflammatory gene expression and inhibits the progression of inflammation. Therefore, the identification of new Nrf2-dependent anti-inflammatory phytochemicals has become a key point in drug discovery. In this review, we discuss the members of the Keap1/Nrf2/ARE signal pathway and its downstream genes, the effects of this pathway on animal models of inflammatory diseases, and crosstalk with the NF-κB pathway. In addition we also discuss about the regulation of NLRP3 inflammasome by Nrf2. Besides this, we summarize the current scenario of the development of anti-inflammatory phytochemicals and others that mediate the Nrf2/ARE signaling pathway [2)].

Breviscapine (BVP) has been widely used in the treatment of several systemic diseases, including those of the cardiovascular and cerebrovascular systems. But, few studies have looked at the neuroprotective effects of BVP and its potential effect in treating traumatic brain injury (TBI). A study of Li et al., from Huai'an, investigated the neuroprotective effect of BVP following traumatic brain injury (TBI) and illuminated the underlying mechanism. The weight drop-induced closed diffuse traumatic brain injury method was used to induce TBI in rats. BVP was injected intraperitoneally 30 minutes after TBI. Neurologic scores were performed to measure behavioral outcomes. Nissl staining and terminal deoxynucleotidyl transferase-mediated dUTP nick-end labeling (TUNEL) assays were performed on histopathologic tissue sections to evaluate neuronal apoptosis. The nuclear factor erythroid 2-related factor 2 (Nrf2) and its related downstream proteins, including heme oxygenase-1 (HO-1) and quinine oxidoreductase-1 (NQO1) were detected with Western blots. BVP treatment alleviated or attenuated TBI-induced neuron cell apoptosis and improved neurobehavioral functions through the upregulated expression of Nrf2 and its related downstream proteins. This study, using the drug, BVP, present new mechanisms responsible for neuronal apoptosis in TBI with possible involvement of the Nrf2 pathway [3)].

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3)
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