## Wogonin

Wogonin is a flavonoid compound with an antioxidant effect extracted from Scutellaria baicalensis Georgi. However, the function and mechanism of wogonin in protecting brain injury remain to be elucidated. A study established a TBI model of Sprague-Dawley rats and treated them with wogonin following trauma. The results showed that wogonin treatment significantly reduced neurobehavioral disorders, brain edema and hippocampal neuron damage caused by TBI. It was found that in TBI rats, administration of wogonin increased the levels of antioxidant factors glutathione, superoxide dismutase and catalase in the CA1 region of the hippocampus and significantly inhibited the production of malondialdehyde and reactive oxygen species. western blotting data showed that wogonin exerted antioxidant activity by downregulating the level of NOX2 protein. In inhibiting cell apoptosis, wogonin upregulated the expression of Bcl-2 protein in the hippocampal CA1 region of TBI rats and inhibited caspase-3 and Bax proteins. Additionally, wogonin inhibited the progression of injury following TBI through the PI3K/Akt/nuclear factor-erythroid factor 2-related factor 2 (Nrf2)/heme oxygenase-1 (HO-1) signaling pathway. Wogonin increased the expression of phosphorylated Akt, Nrf2 and HO-1 in the hippocampus of TBI rats. Following the administration of PI3K inhibitor LY294002, the upregulation of these proteins by wogonin was partly reversed. In addition, LY294002 partially reversed the regulation of wogonin on NOX2, caspase-3, Bax and Bcl-2 proteins. Therefore, wogonin exerts antioxidant and anti-apoptotic properties to prevent hippocampal damage following TBI, which is accomplished through the PI3K/Akt/Nrf2/HO-1 pathway<sup>1)</sup>.

Wogonin might promote the apoptosis of glioma cells by upregulating proapoptotic factors, downregulating antiapoptotic factors, and inhibiting the inflammatory response, thereby inhibiting glioma progression <sup>2</sup>.

Zhuang et al. demonstrated that wogonin dramatically attenuated inflammatory and oxidative stress responses in a murine model of ICH by reducing the expression of pro-inflammatory cytokines and pro-oxidant enzymes such as TNF- $\alpha$ , IL-1 $\beta$ , and inducible nitric oxide synthase (iNOS) after ICH. The effects of wogonin were abolished by administration of the PPAR- $\gamma$  inhibitor GW9662. In conclusion, our data suggest that wogonin facilitates hematoma clearance and neurobehavioral recovery by targeting PPAR- $\gamma$ <sup>3</sup>.

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