## Scutellarin

Scutellarin is a flavone, a type of phenolic chemical compound. It can be found in Scutellaria barbata and S. lateriflora. The determination of the structure of scutellarin took Guido Goldschmiedt many years: after the first publication on that topic in 1901, only in 1910 he managed to obtain enough starting material for more detailed studies.

Scutellarin has been shown to induce apoptosis of ovarian and breast tumor cells in vitro.

Data suggested that scutellarin suppressed metastasis and chemoresistance in glioma cells. Scutellarin might be a new therapeutic approach for the glioma therapy  $^{1)}$ 

It 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, we present new mechanisms responsible for neuronal apoptosis in TBI with possible involvement of the Nrf2 pathway<sup>2</sup>.

Results indicate that Scutellarin could attenuate vasospasm and neurological deficits via modulating the Erk5-KLF2-eNOS pathway after SAH, which may provide an experimental basis for the clinical use of SCU treatment in SAH patients <sup>3)</sup>.

1)

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