

Scutellarin

Catalog No: tcsc4273



Available Sizes

Size: 10mg

Size: 25mg

Size: 50mg



Specifications

CAS No:

27740-01-8

Formula:

$C_{21}H_{18}O_{12}$

Pathway:

PI3K/Akt/mTOR;JAK/STAT Signaling;Stem Cell/Wnt

Target:

Akt;STAT;STAT

Purity / Grade:

>98%

Solubility:

DMSO : ≥ 100 mg/mL (216.28 mM)

Observed Molecular Weight:

462.36

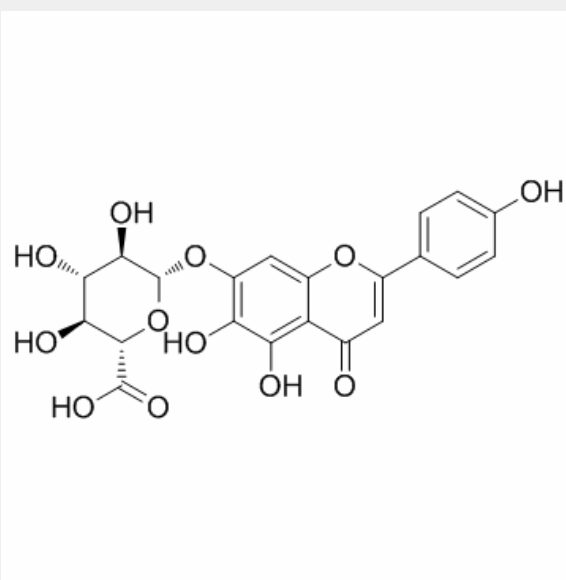
Product Description

Scutellarin, an active flavone isolated from *Scutellaria baicalensis*, can down-regulates the **STAT3/Girdin/Akt** signaling in HCC cells, and inhibits RANKL-mediated **MAPK and NF- κ B** signaling pathway in osteoclasts.

In Vitro:

Scutellarin treatment significantly reduces HepG2 cell viability in a dose-dependent manner, and inhibits migration and invasion of HCC cells in vitro. Scutellarin treatment significantly reduces STAT3 and Girders of actin filaments (Girdin) expression, STAT3 and Akt phosphorylation in HCC cells. Introduction of STAT3 overexpression restores the scutellarin-downregulated Girdin expression, Akt activation, migration and invasion of HCC cells. Furthermore, induction of Girdin overexpression completely abrogates the inhibition of scutellarin on the Akt phosphorylation, migration and invasion of HCC cells. Scutellarin can inhibit HCC cell metastasis in vivo, and migration and invasion in vitro by down-regulating the STAT3/Girdin/Akt signaling^[1]. Scutellarin selectively enhances Akt phosphorylation^[2]. Scutellarin is a putative therapeutic agent as it has been found to not only suppress microglial activation thus ameliorating neuroinflammation, but also enhance astrocytic reaction. Acutellarin amplifies the astrocytic reaction by upregulating the expression of neurotrophic factors among others thus indicating its neuroprotective role. Remarkably, the effects of scutellarin on reactive astrocytes are mediated by activated microglia supporting a functional \"cross-talk\" between the two glial types^[3]. Scutellarin can suppress RANKL-mediated osteoclastogenesis, the function of osteoclast bone resorption, and the expression levels of osteoclast-specific genes (tartrate-resistant acid phosphatase (TRAP), cathepsin K, c-Fos, NFATc1). Further investigation indicates that Scutellarin can inhibit RANKL-mediated MAPK and NF-κB signaling pathway, including JNK1/2, p38, ERK1/2, and IκBα phosphorylation^[5].

In Vivo: Scutellarin (50 mg/kg/day) significantly mitigates the lung and intrahepatic metastasis of HCC tumors in vivo. The numbers of the lung and intrahepatic metastatic tumors in the scutellarin-treated group are significantly less than that in the controls^[1]. The rats treated with Scutellarin display a significant alleviation in neurobehavioral deficits compared to the SAH group. Scutellarin enhanced eNOS expression compared with SAH rats^[4].



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