Scutellarin

Cat. No.: HY-N0751
CAS No.: 27740-01-8
Molecular Formula: C₂₁H₁₈O₁₂
Molecular Weight: 462.36
Target: STAT; Akt
Pathway: JAK/STAT Signaling; Stem Cell/Wnt; PI3K/Akt/mTOR
Storage:
- Powder: -20°C for 3 years, 4°C for 2 years, -80°C for 6 months, -20°C for 1 month
- In solvent: 4°C for 2 years in DMSO

SOLVENT & SOLUBILITY

In Vitro

DMSO: ≥ 100 mg/mL (216.28 mM)

* “≥” means soluble, but saturation unknown.

Preparing Stock Solutions

<table>
<thead>
<tr>
<th>Solvent Concentration</th>
<th>Mass of Solvent</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mg</td>
<td></td>
</tr>
<tr>
<td>1 mM</td>
<td>2.1628 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.4326 mL</td>
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<tr>
<td>10 mM</td>
<td>0.2163 mL</td>
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<tr>
<td>5 mg</td>
<td>10.8141 mL</td>
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<tr>
<td>21.6282 mL</td>
<td></td>
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<tr>
<td>4.3256 mL</td>
<td></td>
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<tr>
<td>2.1628 mL</td>
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</tbody>
</table>

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

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

IC₅₀ & Target

<table>
<thead>
<tr>
<th>STAT3</th>
<th>Akt</th>
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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].

PROTOCOL

Cell Assay [1]

HepG2 cells (1×10^5/well) are cultured in 96-well plates and treated in triplicate with scutellarin at concentrations of 5, 10, 20, 30, and 100 μM or vehicle alone for 24 h. The cellular viability is tested by 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay, and is expressed as a percentage of proliferation versus controls.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Administration [1]

To establish an orthotopic liver xenograft model, individual mice are anesthetized with isoflurane and a small incision is made in their abdomen. Individual mice are injected with 2×10^6 SK-Hep1 cells in 30 μL Matrigel into their left lobe of the liver. Twenty-four hours after orthotopic liver implantation, the mice are randomized and injected intraperitoneally with scutellarin (50 mg/kg/day) or vehicle (0.9% NaCl, normal saline) daily for 35 consecutive days (n=10 per group). Subsequently, the mice are sacrificed, and their lungs and livers are excised, fixed in 10% buffered formalin and paraffin-embedded for hematoxylin and eosin staining.

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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REFERENCES


