### Astragaloside IV

**Cat. No.:** HY-N0431  
**CAS No.:** 84687-43-4  
**Molecular Formula:** C₄₁H₆₈O₁₄  
**Molecular Weight:** 784.97  
**Target:** MMP; ERK; JNK  
**Pathway:** Metabolic Enzyme/Protease; MAPK/ERK Pathway; Stem Cell/Wnt  
**Storage:** Powder  
\[-20^\circ C \text{ 3 years} \]  
\[4^\circ C \text{ 2 years} \]  
\[-80^\circ C \text{ 6 months} \]  
\[-20^\circ C \text{ 1 month} \]

### SOLVENT & SOLUBILITY

**In Vitro**  
DMSO: 390 mg/mL (496.83 mM; Need ultrasonic)

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>Mass (1 mg)</th>
<th>Mass (5 mg)</th>
<th>Mass (10 mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>1.2739 mL</td>
<td>6.3697 mL</td>
<td>12.7393 mL</td>
<td></td>
</tr>
<tr>
<td>5 mM</td>
<td>0.2548 mL</td>
<td>1.2739 mL</td>
<td>2.5479 mL</td>
<td></td>
</tr>
<tr>
<td>10 mM</td>
<td>0.1274 mL</td>
<td>0.6370 mL</td>
<td>1.2739 mL</td>
<td></td>
</tr>
</tbody>
</table>

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

### BIOLOGICAL ACTIVITY

**Description**  
Astragaloside IV, an active component isolated from Astragalus membranaceus, suppresses the activation of ERK1/2 and JNK, and downregulates matrix metalloproteases (MMP)-2, (MMP)-9 in MDA-MB-231 breast cancer cells.

**IC₅₀ & Target**  
MMP-2, MMP-9, ERK1, ERK2, JNK

**In Vitro**  
Astragaloside IV (10, 20, 40 ng/mL) inhibits NSCLC cell growth, whereas low concentrations of astragaloside IV (1, 2.5, 5 ng/mL) has no obvious cytotoxicity on cell viability. Moreover, combined treatment with astragaloside IV significantly increases chemosensitivity to cisplatin in NSCLC cells. On the molecular level, astragaloside IV co-treatment significantly inhibits the mRNA and protein levels of B7-H3 in the presence of cisplatin.[2] Astragaloside IV inhibits the viability and invasive potential of MDA-MB-231 breast cancer cells, suppresses the activation of the mitogen activated protein kinase (MAPK) family members ERK1/2 and JNK, and downregulates matrix metalloproteases (MMP)-2 and -9.[4]
Astragaloside IV (10, 20 mg/kg, p.o.) exhibits a potent ability to prevent cognitive deficits induced by transient cerebral ischemia and reperfusion. Astragaloside IV (10 mg/kg) and Astragaloside IV (20 mg/kg) can significantly decrease the levels of these cytokines compared to the Model group. Astragaloside IV significantly inhibits the level of TLR4 and its downstream proteins, suggesting that both MyD88-dependent and -independent pathways play important roles in the anti-inflammatory effects of Astragaloside IV. Astragaloside IV attenuates NLRP3 and cleaved-caspase-1 expression, and reduces Iba1 protein expression[1]. In the mice model, the high-dose astragaloside IV group has a significant increase in the 48-hour survival rate [60% (9/15) vs 13.3% (2/15), P < 0.05], significant reductions in the serum ALT and AST levels (P < 0.01), and significant reductions in liver histopathological indices and the degree of apoptosis of hepatocytes (P < 0.01), as well as a significant reduction in the content of MDA in liver homogenate (P < 0.01) and a significant increase in the activity of SOD[3].

**PROTOCOL**

**Kinase Assay [4]**

Briefly, MDA-MB-231 cells treated as indicated or tumor tissues are harvested and lysed in Mg$^{2+}$ lysis buffer containing 50 mM Tris (pH 7.5), 10 mM MgCl$_2$, 0.5 M NaCl, and protease inhibitor cocktail. Equal amounts of lysates are incubated with PAK-PBD beads at 4°C for 1 h. PAK-PBD beads are pelleted by centrifugation and washed with ish buffer containing 25 mM Tris (pH 7.5), 30 mM MgCl$_2$, 40 mM NaCl. Active Rac1 is detected by western blotting. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**Cell Assay [2]**

Cell viability is determined by CCK-8 assay. To be brief, cultured NSCLC cells are seeded into 96-well plates at the density of 4×10$^4$ (cells/well). Then 10 µL/well CCK8 solution is added and incubated in dark at 37°C for another 2 h. The absorbance is determined with the wavelength of 490 nm. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

**Animal Administration [1]**

Transient cerebral ischemia and reperfusion is prepared by BCCAO, as BCCAO is considered an ideal model to study transient cerebral ischemia and reperfusion injury-mediated inflammatory response. Mice are randomly divided into the Sham, Model, Astragaloside IV (10 mg/kg) and Astragaloside IV (20 mg/kg) treatment groups. The Astragaloside IV treatment groups are intragastrically administered 7 days before the surgery and terminated on the day of sacrifice. On the day of the surgery, Astragaloside IV is administrated 2 h prior to ischemia. The Sham-operated and Model groups are treated with distilled water. After the mice are anesthetized with an intraperitoneal injection of chloral hydrate (350 mg/kg), the bilateral common carotid arteries are exposed and carefully separated with a small ventral neck incision and occluded twice (20 min each) with ligated surgical silk as described previously with minor modifications. There is a 10 min reperfusion period between the two occlusion periods (ischemia 20 min – reperfusion 10 min – ischemia 20 min). Sham-operated mice are subjected to the same surgical operation without the surgical silk ligation. Mouse body temperature is maintained at 37±0.5°C during the surgery with heating equipment until recovery from the anesthesia. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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**REFERENCES**

[1]. Li M, et al. Astragaloside IV attenuates cognitive impairments induced by transient cerebral ischemia and reperfusion in mice via anti-inflammatory

