Astragaloside IV

Cat. No.: HY-N0431

CAS No.: 84687-43-4

Molecular Formula: $C_{41}H_{68}O_{14}$

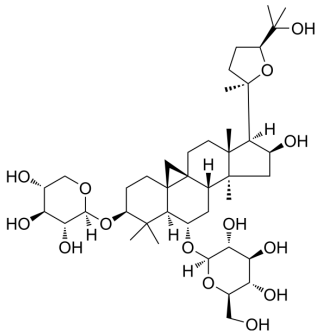
Molecular Weight: 784.97

Target: MMP; ERK; JNK

Pathway: Metabolic Enzyme/Protease; MAPK/ERK Pathway; Stem Cell/Wnt

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (127.39 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.2739 mL	6.3697 mL	12.7393 mL
5 mM	0.2548 mL	1.2739 mL	2.5479 mL
10 mM	0.1274 mL	0.6370 mL	1.2739 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.5 mg/mL (3.18 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.18 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.18 mM); Clear solution

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.

IC_{50} & 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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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.

CUSTOMER VALIDATION

- Int Immunopharmacol. 2020 Dec;89(Pt A):107169.

- Front Pharmacol. 2018 Apr 16;9:345.
- Eur J Pharmacol. 2020 Oct 15;885:173399.
- Bioengineered. 2022 Apr;13(4):8240-8254.
- Heliyon. 2023 Apr 11.

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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 mechanisms. Neurosci Lett. 2016 Dec 20. pii: S0304-3940(16)30994-

[2]. He CS, et al. Astragaloside IV Enhances Cisplatin Chemosensitivity in Non-Small Cell Lung Cancer Cells Through Inhibition of B7-H3. Cell Physiol Biochem. 2016;40(5):1221-1229. Epub 2016 Dec 14.

[3]. Liu L, et al. [Protective effect of astragaloside IV against acute liver failure in experimental mice]. Zhonghua Gan Zang Bing Za Zhi. 2016 Oct 20;24(10):772-777

[4]. Jiang K, et al. Astragaloside IV inhibits breast cancer cell invasion by suppressing Vav3 mediated Rac1/MAPK signaling. Int Immunopharmacol. 2016 Dec 5;42:195-20

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