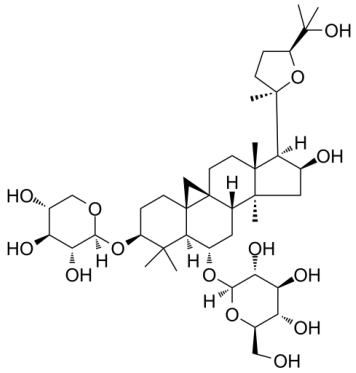
Data Sheet

Product Name:	Astragaloside IV
Cat. No.:	CS-4272
CAS No.:	84687-43-4
Molecular Formula:	$C_{41}H_{68}O_{14}$
Molecular Weight:	784.97
Target:	ERK; JNK; MMP
Pathway:	MAPK/ERK Pathway; Metabolic Enzyme/Protease; Stem Cell/Wnt
Solubility:	DMSO : ≥ 100 mg/mL (127.39 mM)



BIOLOGICAL ACTIVITY:

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. **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]. **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].

PROTOCOL (Extracted from published papers and Only for reference)

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. **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. **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.

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

CAIndexNames:

β-D-Glucopyranoside, (3β,6α,16β,20R,24S)-20,24-epoxy-16,25-dihydroxy-3-(β-D-xylopyranosyloxy)-9,19-cyclolanostan-6-yl

SMILES:

O[C@H]1[C@H](O)[C@@H](O)[C@]([H])(O[C@@H]2C(C)(C)[C@@]([C@@H](O[C@]3([H])O[C@H](CO)[C@@H](O)[C@H](O)[C@H]3O)C[C@]4([H])[C@@]56CC[C@@]7(C)[C@@]4(C)C[C@H](O)[C@]7([H])[C@]8(C)O[C@H](C(C)(O)C)CC8)([H])[C@]5(C6)CC2)OC1

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