Astragaloside VI

Cat. No.: HY-N6577

CAS No.: 84687-45-6

Molecular Formula: $C_{47}H_{78}O_{19}$

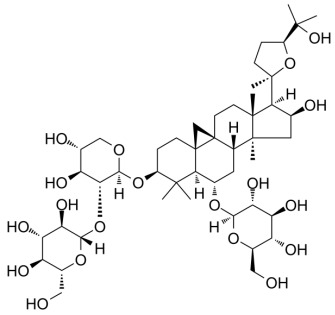
Molecular Weight: 947.11

Target: EGFR

Pathway: JAK/STAT Signaling; Protein Tyrosine Kinase/RTK

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 (105.58 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.0558 mL	5.2792 mL	10.5584 mL
5 mM	0.2112 mL	1.0558 mL	2.1117 mL
10 mM	0.1056 mL	0.5279 mL	1.0558 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 (2.64 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (2.64 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (2.64 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Astragaloside VI could activate EGFR/ERK signalling pathway to improve wound healing.

IC_{50} & Target

EGFR

In Vitro

Pretreatment with Astragaloside VI (AS-VI) at 1 μM increases EGFR activation in HaCaT cells. Astragaloside VI, a major intestinal metabolite of astragalosides, exerts the strongest EGFR activation. In HaCaT cells, the positive control, EGF expectedly results in 1.5±0.03-fold increase in cell proliferation, compared to the control. Astragaloside VI at the indicated

	concentrations also significantly promots cell proliferation in both HaCaT and HDF cells[1]. Astragaloside VI promotes neural stem cell proliferation and enhances neurological function recovery in transient cerebral ischemic injury via activating EGFR/MAPK signaling cascades[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Astragaloside VI improves wound healing, compared to the control. In the simple noninfected wound model, wound healing in mice is accelerated by Astragaloside VI, where in the time required for wound closure is shortened by approximately 2-4 days, compared to that in the control group. Topical treatment with Astragaloside VI reduces the volume of pus produced, compared to the control group. Astragaloside VI treated wounds show an accelerated rate of healing, compared to the control and vaseline groups. By day 22, the Astragaloside VI -treated wounds fully close, whereas the blank and vaseline-treated wounds do not fully close until day 26. Angiogenesis is a crucial step in the formation of granulation tissue and wound healing. Astragaloside VI increases blood vessel formation in both the non-infected and infected wound models[1]. Astragaloside VI could effectively activate EGFR/MAPK signaling cascades, promote NSCs proliferation and neurogenesis in transient cerebral ischemic brains, and improve the repair of neurological functions in post-ischemic stroke rats[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Lee SY, et al. Astragaloside VI and cycloastragenol-6-O-beta-D-glucoside promote wound healing in vitro and in vivo. Phytomedicine. 2018 Jan 1;38:183-191.

[2]. Chen X, et al. Astragaloside VI Promotes Neural Stem Cell Proliferation and Enhances Neurological Function Recovery in Transient Cerebral Ischemic Injury via Activating EGFR/MAPK Signaling Cascades. Mol Neurobiol. 2018 Aug 7.

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