

Product Data Sheet

Product Name: astragalin

Cat. No.: GN10561

Chemical Properties

Cas. No. 480-10-4

Chemical Name 5,7-dihydroxy-2-(4-hydroxyphenyl)-3-[(2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)oxan-2-yl]oxochromen-4-one

SMILES C1=CC(=CC=C1C2=C(C(=O)C3=C(C=C(C=C3O2)O)O)OC4C(C(C(C(O4)CO)O)O)O)O

Formula $C_{21}H_{20}O_{11}$

M.Wt 448.38

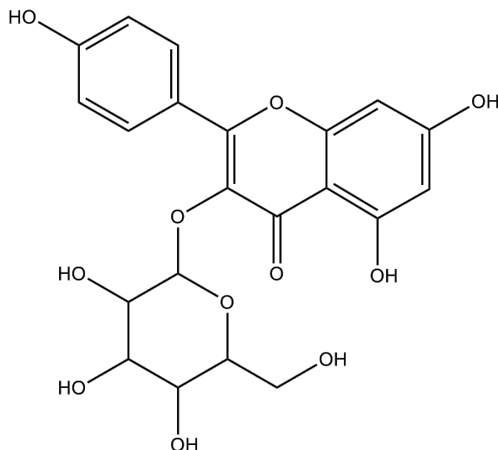
Solubility $\geq 44.8\text{mg/mL}$ in DMSO

Storage Store at 2-8°C, protect from light

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines BEAS-2B cells

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

Preparation Method BEAS-2B cells were seeded into 6-well plates at a density of 5×10^4 cells per well and cultured in HEPES-buffered M199 medium containing 10% FBS and other supplements for 24 hours. The cells were then pretreated with different concentrations (1, 5, 10, and 20 μ M) of Astragalin for 1 hour before being stimulated with 20 μ M H₂O₂ for up to 72 hours.

Reaction Conditions 1, 5, 10, and 20 μ M Astragalin; 20 μ M H₂O₂; 72 hours

Applications Astragalin significantly restored the expression of E-cadherin and inhibited the induction of vimentin in H₂O₂-treated BEAS-2B cells, indicating its ability to attenuate EMT. Astragalin blocked the induction of autophagy-related proteins beclin-1 and LC3A/B and reduced autophagosome formation.

Animal experiment [2]:

Animal models C57BL/6J mice

Preparation Method Six-week-old male C57BL/6J mice were used. Diabetes was induced by intraperitoneal injection of streptozotocin (STZ, 100mg/kg/day) for two consecutive days. Seven days after injection, mice with random blood glucose ≥ 16.7 mmol/L were randomly divided into groups and treated with Astragalin (5 and 10mg/kg/day) by oral gavage for 4 weeks. At the end of the experiment, blood and kidney tissues were collected for analysis.

Dosage form 5 and 10mg/kg/day for 4 weeks; p.o.

Applications Astragalin significantly improved renal function by reducing proteinuria, lowering serum creatinine levels, and improving kidney pathological damage in diabetic mice. It also inhibited aldose reductase activity, reduced oxidative stress, improved mitochondrial dysfunction, and regulated mitochondrial dynamics by activating the AMPK/PGC1 α pathway.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

References:

- [1] Sun MY, Ye HJ, Zheng C, et al. Astragalin ameliorates renal injury in diabetic mice by modulating mitochondrial quality control via AMPK-dependent PGC1 α pathway. *Acta Pharmacol Sin.* 2023 Aug;44(8):1676-1686.
- [2] Sun MY, Ye HJ, Zheng C, et al. Astragalin ameliorates renal injury in diabetic mice by modulating mitochondrial quality control via AMPK-dependent PGC1 α pathway. *Acta Pharmacol Sin.* 2023 Aug;44(8):1676-1686.

Background

Astragalin is a natural flavonoid compound with strong antioxidant and anti-inflammatory activities and has shown potential therapeutic applications in various diseases, including cancer and cardiovascular diseases^[1]. Astragalin is extracted from plants such as Mongolian milkvetch (*Astragalus membranaceus*) and sumac (*Rhus chinensis*)^[2]. In cancer research, Astragalin has been reported to inhibit the proliferation of cancer cells and induce apoptosis^[3]. Astragalin also protects the cardiovascular system by reducing oxidative stress and inflammation^[4].

In vitro, Astragalin (0–100 μ M) treatment of human colorectal epithelial cells (HCT-116 and HT-29) significantly inhibited cell proliferation. Pre-treatment with Astragalin significantly reduced the mRNA expression levels of tumor necrosis factor- α (TNF- α)-induced interleukin-6 (IL-6), IL-8, and TNF- α . Additionally, Astragalin inhibited the phosphorylation and degradation of I κ B α induced by TNF- α and reduced the DNA-binding activity of NF- κ B^[5]. Astragalin (1–20 μ M) treatment of human bronchial epithelial cells (BEAS-2B) significantly inhibited the epithelial-mesenchymal transition (EMT) process induced by oxidative stress. Astragalin restored the reduced expression of E-cadherin induced by H₂O₂ and dose-dependently inhibited the expression of vimentin and the formation of autophagosomes^[6].

In vivo, Astragalin (10, 20, 40mg/kg) was administered via intraperitoneal injection to APP/PS1 transgenic mice once daily for one month. Astragalin significantly improved cognitive dysfunction in APP/PS1 mice. Astragalin also alleviated neuronal damage in the hippocampus, reduced amyloid- β (A β) deposition and the number of senile plaques, and activated autophagy by promoting the expression of autophagy-related proteins (LC3B, Beclin-1, ATG5, ATG12, and LAMP-1). Furthermore, Astragalin inhibited the phosphorylation levels of PI3K/Akt-mTOR signaling pathway-

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

Product Data Sheet

related proteins, thereby exerting neuroprotective effects^[7]. Astragalín (5, 10mg/kg/day) was administered orally to streptozotocin (STZ)-induced diabetic mice for 4 weeks. Astragalín significantly improved proteinuria and reduced serum creatinine levels in diabetic mice. Astragalín also alleviated renal pathological damage. Astragalín significantly inhibited the activity of aldose reductase (ALR2) in the kidneys, reduced oxidative stress, improved mitochondrial dysfunction, promoted mitochondrial biogenesis, maintained mitochondrial dynamic balance, and reduced renal cell apoptosis by activating the AMPK-dependent PGC1 α pathway^[8].

References:

- [1] Chen J, Zhong K, Qin S, et al. Astragalín: a food-origin flavonoid with therapeutic effect for multiple diseases. *Front Pharmacol*. 2023 Oct 18;14:1265960.
- [2] Riaz A, Rasul A, Hussain G, et al. Astragalín: A Bioactive Phytochemical with Potential Therapeutic Activities. *Adv Pharmacol Sci*. 2018 May 2;2018:9794625.
- [3] Ruan J, Shi Z, Cao X, et al. Research Progress on Anti-Inflammatory Effects and Related Mechanisms of Astragalín. *Int J Mol Sci*. 2024 Apr 19;25(8):4476.
- [4] Zhang Q, Yan Y. The role of natural flavonoids on neuroinflammation as a therapeutic target for Alzheimer's disease: a narrative review. *Neural Regen Res*. 2023 Dec;18(12):2582-2591.
- [5] Han YM, Koh J, Kim JH, et al. Astragalín Inhibits Nuclear Factor- κ B Signaling in Human Colonic Epithelial Cells and Attenuates Experimental Colitis in Mice. *Gut Liver*. 2021 Jan 15;15(1):100-108.
- [6] Cho IH, Choi YJ, Gong JH, et al. Astragalín inhibits autophagy-associated airway epithelial fibrosis. *Respir Res*. 2015 Apr 21;16(1):51.
- [7] Yang CZ, Wang SH, Zhang RH, et al. Neuroprotective effect of astragalín via activating PI3K/Akt-mTOR-mediated autophagy on APP/PS1 mice. *Cell Death Discov*. 2023 Jan 21;9(1):15.
- [8] Sun MY, Ye HJ, Zheng C, et al. Astragalín ameliorates renal injury in diabetic mice by modulating mitochondrial quality control via AMPK-dependent PGC1 α pathway. *Acta Pharmacol Sin*. 2023 Aug;44(8):1676-1686.

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA