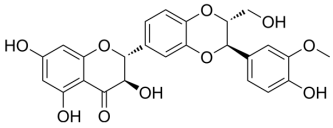
Silybin A

Cat. No.:	HY-13748
CAS No.:	22888-70-6
Molecular Formula:	$C_{25}H_{22}O_{10}$
Molecular Weight:	482.44
Target:	Autophagy; Reactive Oxygen Species
Pathway:	Autophagy; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB
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 : 250 mg/mL (518.20 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0728 mL	10.3640 mL	20.7280 mL
	5 mM	0.4146 mL	2.0728 mL	4.1456 mL
	10 mM	0.2073 mL	1.0364 mL	2.0728 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 (5.18 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.18 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.18 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Silybin A (Silibinin A), an effective anti-cancer and chemopreventive agent, has been shown to exert multiple effects on cancer cells, including inhibition of both cell proliferation and migration.

In Vitro

Silybin A (Silybin) significantly induced the expression of the non-steroidal anti-inflammatory drug-activated gene-1 (NAG-1) in both p53 wild-type and p53-null cancer cell lines[1].
Silybin A (Silybin) induced cell death in human breast cancer cell lines MCF7 and MDA-MB-231[2].
Silybin A (Silybin) treatment resulted in a dose- and time-dependent inhibition of HCC cell viability[3].

	Silybin A (Silybin) treatment decreased the expression of the Notch1 intracellular domain (NICD), RBP-Jκ, and Hes1 proteins, upregulated the apoptosis pathway-related protein Bax, and downregulated Bcl2, survivin, and cyclin D1[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Topical application of Silybin A (Silibinin A) at the dose of 9 mg/mouse effectively suppressed oxidative stress and deregulated activation of inflammatory mediators and tumorigenesis[4]. The kidney cortex of vehicle-treated control OVE26 mice displayed greater Nox4 expression and twice as much superoxide production than cortex of silybin-treated mice. The glomeruli of control OVE26 mice displayed 35% podocyte drop out that was not present in the silybin-treated mice[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Microbiome. 2019 Mar 20;7(1):43.
- Acta Pharm Sin B. 2021 Jan;11(1):143-155.
- Cell Death Dis. 2020 Aug 14;11(8):630.
- Phytomedicine. 21 July 2022, 154349.
- Viruses. 2020 Jun 10;12(6):628.

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REFERENCES

[1]. Woo SM, et al. Silibinin induces apoptosis of HT29 colon carcinoma cells through early growth response-1 (EGR-1)-mediated non-steroidal anti-inflammatory drug-activated gene-1 (NAG-1) up-regulation. Chem Biol Interact. 2014 Jan 16;211C:36-43.

[2]. Kim TH, et al. Silibinin induces cell death through ROS-dependent down-regulation of Notch-1/ERK/Akt signaling in human breast cancer cells. J Pharmacol Exp Ther. 2014 Jan 28.

[3]. Zhang S, et al. Silybin-mediated inhibition of Notch signaling exerts antitumor activity in human hepatocellular carcinoma cells. PLoS One. 2013 Dec 27;8(12):e83699.

[4]. Khan AQ, et al. Silibinin Inhibits Tumor Promotional Triggers and Tumorigenesis Against Chemically Induced Two-Stage Skin Carcinogenesis in Swiss Albino Mice: Possible Role of Oxidative Stress and Inflammation. Nutr Cancer. 2013 Dec 23.

[5]. Khazim K, et al. The antioxidant silybin prevents high glucose-induced oxidative stress and podocyte injury in vitro and in vivo. Am J Physiol Renal Physiol. 2013 Sep 1;305(5):F691-700.

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