Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Silibinin

Catalog No: tcsc2128

Available Sizes

Size: 100mg

Size: 500mg

Specifications

CAS No:
22888-70-6

Formula:
$C_{25}H_{22}O_{10}$

Pathway:
Autophagy

Target:
Autophagy

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 31 mg/mL (64.26 mM)

Alternative Names:
Silybin;Silibinin A;Silymarin I

Observed Molecular Weight:
482.44

Product Description

Silibinin, 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.

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IC50 value:

Target: anticancer

in vitro: silibinin 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, suggesting that silibinin-induced NAG-1 up-regulation is p53-independent manner.Silibinin up-regulates early growth response-1 (EGR-1) expression [1]. silibinin induced cell death in human breast cancer cell lines MCF7 and MDA-MB-231. Silibininduced cell death was attenuated by antioxidants, N-acetylcysteine (NAC) and Trolox, suggesting that the effect of silibinin was dependent on generation of reactive oxygen species (ROS) [2]. SIL treatment resulted in a dose- and time-dependent inhibition of HCC cell viability, SIL exhibited strong antitumor activity, as evidenced not only by reductions in tumor cell adhesion, migration, intracellular glutathione (GSH) levels and total antioxidant capability (T-AOC) but also by increases in the apoptotic index, caspase3 activity, and reactive oxygen species (ROS). SIL 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. Notch1 siRNA (in vitro) or DAPT (a known Notch1 inhibitor, in vivo) further enhanced the antitumor activity of SIL, and recombinant Jagged1 protein (a known Notch ligand in vitro) attenuated the antitumor activity of SIL [3].

in vivo: Topical application of silibinin 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].