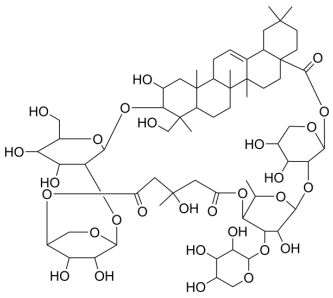
Tubeimoside I

Cat. No.:	HY-N0890		
CAS No.:	102040-03-9		
Molecular Formula:	$C_{63}H_{98}O_{29}$		
Molecular Weight:	1319.44		
Target:	Apoptosis		
Pathway:	Apoptosis		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (75.79 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.7579 mL	3.7895 mL	7.5790 mL
	5 mM	0.1516 mL	0.7579 mL	1.5158 mL
	10 mM	0.0758 mL	0.3789 mL	0.7579 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 (1.89 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (1.89 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (1.89 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Tubeimoside I(Lobatoside-H) is an extract from Chinese herbal medicine Bolbostemma paniculatum (MAXIM.) FRANQUET (Cucurbitaceae) has been shown as a potent anti-tumor agent for a variety of human cancers.IC50 value:Target: Anticancer natural compoundin vitro: TBMS I inhibited the proliferation of both HepG2 and L-02 cells in a dose- and time-dependent manner, but HepG2 cells appeared more sensitive to the agent. When exposed to TBMS I for 24, 48 and 72 h, IC50 for HepG2 cells versus L-02 cells were 15.5 vs. 23.1, 11.7 vs. 16.2, 9.2 vs. 13.1 (μM, $p<0.01$), respectively. TBMS I induced cell shrinkage, nuclear condensation and fragmentation, cell cycle arrest at the G2/M phase, mitochondrial membrane disruption, release of cytochrome c from the mitochondria, activation of caspase 3 and 9, and shifting Bax/Bcl-2 ratio from being anti-apoptotic

to pro-apoptotic, all indicative of initiation and progression of apoptosis involving mitochondrial dysfunction [1]. TBMS1-induced molecular events were related to mitochondria-induced intrinsic apoptosis and P21-cyclin B1/cdc2 complex-related G2/M cell cycle arrest [2]. TBMS1 combined with CDDP promoted cell apoptosis, decreased proliferation activity and increased cytosolic Ca2+ levels. Bcl-2 protein expression was down-regulated but Bax was up-regulated. Moreover, GST-π mRNA and protein expression were decreased. TBMS1 reduced the resistance of the cells to CDDP-induced cytotoxicity [4]. Treatment with TBMS1 resulted in dose- and time-dependent inhibition of proliferation, led to arrest in phase G2/M of the cell cycle and increased the levels of intracellular Ca2 . Furthermore, TBMS1 up-regulated the levels of the glucose-regulated protein 78/immunoglobuin heavy chain binding protein (GRP78/Bip), C/EBP homologous protein (CHOP), Bax, and cleaved caspase-3 and down-regulated the levels of Bcl-2 [5].in vivo: TBMS1 significantly inhibited the production of the pro-inflammatory cytokines, TNF-α, IL-6 and IL-1β in vitro and in vivo. Pretreatment with TBMS1 markedly attenuated the development of pulmonary edema, histological severities and inflammatory cells infiltration in mice with ALI [3].

CUSTOMER VALIDATION

- Pharmacol Res. 2020 May;155:104751.
- Oxid Med Cell Longev. 2023 Jan 14;2023:9966355.

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REFERENCES

[1]. Wang Y, et al. Natural plant extract tubeimoside I promotes apoptosis-mediated cell death in cultured human hepatoma (HepG2) cells. Biol Pharm Bull. 2011;34(6):831-8.

[2]. Xu Y, et al. Intrinsic apoptotic pathway and G2/M cell cycle arrest involved in tubeimoside I-induced EC109 cell death. Chin J Cancer Res. 2013 Jun;25(3):312-21.

[3]. Wu Q, et al. Tubeimoside-1 attenuates LPS-induced inflammation in RAW 264.7 macrophages and mouse models. Immunopharmacol Immunotoxicol. 2013 Aug;35(4):514-23.

[4]. Liu HZ, et al. Tubeimoside I sensitizes cisplatin in cisplatin-resistant human ovarian cancer cells (A2780/DDP) through down-regulation of ERK and up-regulation of p38 signaling pathways. Mol Med Rep. 2011 Sep-Oct;4(5):985-92.

[5]. Chen WJ, et al. Tubeimoside-1 induces G2/M phase arrest and apoptosis in SKOV-3 cells through increase of intracellular Ca2+ and caspase-dependent signaling pathways. Int J Oncol. 2012 Feb;40(2):535-43.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA