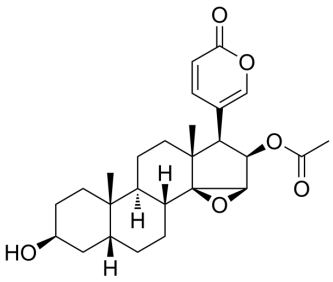
Cinobufagin

Cat. No.:	HY-N0421		
CAS No.:	470-37-1		
Molecular Formula:	$C_{26}H_{34}O_6$		
Molecular Weight:	442.54		
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 (225.97 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2597 mL	11.2984 mL	22.5968 mL
	5 mM	0.4519 mL	2.2597 mL	4.5194 mL
	10 mM	0.2260 mL	1.1298 mL	2.2597 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 (5.65 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.65 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.65 mM); Clear solution
4. Add each solvent one by one: 0.5% β-Cyclodextrin in Saline
   Solubility: ≥ 0.97 mg/mL (2.19 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cinobufagin is an anticancer agent that can be secreted by the Asiatic toad Bufo gargarizans. Cinobufagin induces the cell cycle arrests in the G1 phase or G2/M phase, leading to apoptosis in cancer cells. Cinobufagin inhibits tumor growth in melanoma and glioblastoma multiforme xenograft mouse models[1][2][3].

In Vitro

Conobufagin (30-300 nM, 7 days) exerts potent antitumor activity in a dose-dependent manner in uveal melanoma OCM1 cells[1].
Conobufagin (30-300 nM, 24 hours) arrests the cell cycle in the G1 phase in a concentrationdependent manner and induces cell apoptosis and alterations of apoptosis-related proteins in OCM1 cells[1].
Conobufagin (0.01-1 μM, 6 hours) blocks EGFR phosphorylation and induces cell apoptosis and cytotoxicity in glioblastoma multiforme U87MG-EGFR and U87MG-PTEN cells[2].
Cinobufagin (0.4,0.7,1.0 μM, 24-48 hours) induces cell cycle arrest at the G2/M phase and cell apoptosis, leading to inhibition of malignant melanoma A375/B16 cell proliferation[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	OCM1 cell
Concentration:	30,100,300 nM
Incubation Time:	7 days
Result:	Exerted potent cytotoxic in OCM1 cells with an IC_{50} of 8.023 nM.

Apoptosis Analysis[1]

Cell Line:	OCM1 cell
Concentration:	30,100,300 nM
Incubation Time:	24 hours
Result:	Induced cell apoptosis and upregulate the expression levels of cleaved caspase-3, cleaved poly(ADP-ribose) polymerase (PARP), and cleaved caspase-9. Activated the intrinsic mitochondrial apoptosis pathway, which was demonstrated by increased cell apoptosis with increased expression of Bad and Bax, decreased expression of Bcl-2 and Bcl-xl, and reduced mitochondrial membrane potential (MMP).

In Vivo

Cinobufagin (5 mg/kg for i.p., once a day for 10 days) inhibits xenograft growth by inducing cell apoptosis in tumor xenograft mice model[1].
Cinobufagin (5 mg/kg for i.p., once a day for 10 days) suppresses tumor growth in both subcutaneous and intracranial U87MG-EGFR xenograft mouse models and increases the median survival of nude mice bearing intracranial U87MGEGFR tumors[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	OCM1 cells tumor xenograft in Nu/Nu nude mice [1]
Dosage:	5 mg/kg
Administration:	Intraperitoneal injection (i.p.), once a day for 10 days
Result:	Made the tumors grew more slowly than those treated with intraperitoneal injection of saline or untreated. Increased the expression of caspase-3 and PARP in tumor tissues and decreased Bcl-2 and Bcl-xl expression in mouse tumor tissues and increased expression of Bad and Bax.

Animal Model:	U87MG-EGFR subcutaneous and intracranial xenograft model[2]
Dosage:	5 mg/kg

Administration:	Intraperitoneal injection (i.p.), once a day for 10 days
Result:	Decreased the luminescence intensity of brain tumor about 70%. Decreased p-EGFR, p-STAT3, and p-Akt levels in the intracranial tumors as compared with the vehicles. Decreased Ki67 and active caspase-3 immunostaining of intracranial tumors.

CUSTOMER VALIDATION

- Exp Cell Res. 2020 Aug 1;393(1):112054.
- Research Square Preprint. 2023 Jul 18.

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REFERENCES

[1]. Guangxin Zhang, et al. Cinobufagin inhibits tumor growth by inducing intrinsic apoptosis through AKT signaling pathway in human nonsmall cell lung cancer cells. Oncotarget. 2016 Mar 3.

[2]. Yang Yu, et al. Immunomodulatory Effects of Cinobufagin on Murine Lymphocytes and Macrophages. Evid Based Complement Alternat Med. 2015;2015:835263.

[3]. Baek SH, et al. Cinobufagin exerts anti-proliferative and pro-apoptotic effects through the modulation ROS-mediated MAPKs signaling pathway. Immunopharmacol Immunotoxicol. 2015 Jun;37(3):265-73.

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