cRIPGBM chloride

Cat. No.: HY-115630

CAS No.: 2361988-77-2

Molecular Formula: $C_{26}H_{20}ClFN_2O_2$

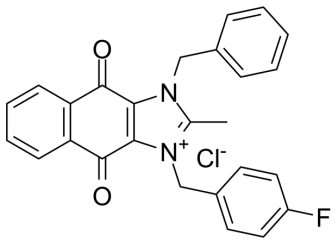
Molecular Weight: 446.9

Target: Caspase; Apoptosis; RIP kinase

Pathway: Apoptosis

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (55.94 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2376 mL	11.1882 mL	22.3764 mL
	5 mM	0.4475 mL	2.2376 mL	4.4753 mL
	10 mM	0.2238 mL	1.1188 mL	2.2376 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

cRIPGBM chloride, an orally active, proapoptotic derivative. cRIPGBM can be generated from glioblastoma multiforme (GBM) cancer stem cells (CSCs). cRIPGBM(chloride) targets to receptor-interacting protein kinase 2 (RIPK2) to induce caspase 1-dependent apoptosis. cRIPGBM(chloride) suppresses the formation of RIPK2/TAK1 (prosurvival complex), and increases the formation of RIPK2/caspase 1 (proapoptotic complex). cRIPGBM(chloride) exerts potent anti-tumor activity in vivo in animal models[1].

IC_{50} & Target	Caspase-1	RIPK2

In Vitro

cRIPGBM chloride (0.25 μM; 0-24 h) time-dependently activates caspase 1, caspase 9, and caspase 7, as well as PARP cleavage, in CBM-1 GBM CSCs[1].

cRIPGBM chloride (0.125 μM, 0.25 μM; 24 h) induces cell apoptosis mediated by caspase 1 in CBM-1 GBM CSCs[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	GBM-1 GBM CSCs

Concentration:	50 nM, 100 nM, 125 nM, 250 nM, and 500 nM
Incubation Time:	3 h, 6 h, 12 h, and 24 h
Result:	Had the ability to regulate RIPK2 to act as a prosurvival or proapoptotic molecule. Significantly reduced RIPK2 binding to cIAP2 in a dose-dependent manner.

In Vivo

cRIPGBM chloride (50 mg/kg; p.o.; twice daily for 5 weeks) inhibits tumor growth in patient-derived GBM CSC intracranial xenograft mouse models[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Orthotopic intracranial xenograft model in mouse[1]
Dosage:	50 mg/kg
Administration:	PO; twice daily, 8 h apart, starting at day 7 postinjection; last for 5 weeks
Result:	Monitored by Fluorescence Tomography System. Decreased the tumor signal, as well as tumor size.

REFERENCES

[1]. Lucki NC, et al. A cell type-selective apoptosis-inducing small molecule for the treatment of brain cancer. Proc Natl Acad Sci U S A. 2019 Mar 26;116(13):6435-6440.

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