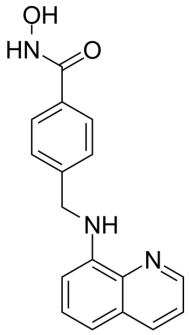
MPT0G211

Cat. No.:	HY-123976
CAS No.:	2151853-97-1
Molecular Formula:	$C_{17}H_{15}N_3O_2$
Molecular Weight:	293.32
Target:	HDAC
Pathway:	Cell Cycle/DNA Damage; Epigenetics
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 : 100 mg/mL (340.92 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4092 mL	17.0462 mL	34.0925 mL
	5 mM	0.6818 mL	3.4092 mL	6.8185 mL
	10 mM	0.3409 mL	1.7046 mL	3.4092 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.52 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

MPT0G211 is a potent, orally active and selective HDAC6 inhibitor (IC_{50}=0.291 nM). MPT0G211 displays >1000-fold selective for HDAC6 over other HDAC isoforms. MPT0G211 can penetrate the blood-brain barrier. MPT0G211 ameliorates tau phosphorylation and cognitive deficits in an Alzheimer's disease model. MPT0G211 has anti-metastatic and neuroprotective effects. Anticancer activities[1][2][3].

IC_{50} & Target

HDAC6
0.291 μM (IC_{50})

In Vitro

MPT0G211 (0.1 μM; cells were transfected with pCAX APP 695 and pRK5-EGFP-Tau P301L for 24 h) significantly inhibits the phosphorylation of tau Ser396[1].
MPT0G211 inhibits HDAC6/Hsp90 binding and causes subsequent proteasomal degradation of polyubiquitinated proteins[1].
MPT0G211 significantly decreases the phosphorylation of tau by GSK3β inactivation[1].

MPT0G211 (0.1 μM; 24 hours) significantly attenuates the phosphorylation of tau Ser396 and Ser404 in both cell lines (SH-SY5Y and Neuro-2a cells were transfected for 24 h with pCAX APP 695 and pRK5-EGFP-Tau P301L)[1].
MPT0G211 inhibits MDA-MB-231 and MCF-7 cells growth (GI_{50}=16.19 and 5.6 μM, respectively)[2].
In AML cells, MPT0G211 potentiated the cytotoxic effects of DOXO by impairing DNA repair machinery and activating Bcl-2-associated X protein (BCL-XL)-dependent cell apoptosis[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

MPT0G211 (50 mg/kg; p.o.; daily for 3 months) significantly ameliorates the spatial memory impairment[1].
MPT0G211 (25 mg/kg; i.p. ; qd; day 73 post-tumor injection) reduces numbers of nodules and lung weights[2].
MPT0G211 treatment not only diminishes tau phosphorylation by inhibition GSK3β activity but also enhances the acetylation of Hsp90, which causes the downregulation of HDAC6/Hsp90 binding and facilitates proteasomal degradation of polyubiquitinated p-tau[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Triple transgenic (3×Tg-AD) mice (harboring APP_{Swe} and tau_{P301L} mutant transgenes[1]
Dosage:	50 mg/kg
Administration:	P.o.; daily for 3 months
Result:	Significantly ameliorated the spatial memory impairment.

Animal Model:	Female SCID mice (bearing MDA-MB-231 cells)[2]
Dosage:	25 mg/kg
Administration:	I.p.; qd; day 73 post-tumor injection
Result:	Significantly reduced numbers of nodules and lung weights.

REFERENCES

[1]. Fan SJ, Huang FI, et al. The novel histone de acetylase 6 inhibitor, MPT0G211, ameliorates tau phosphorylation and cognitive deficits in an Alzheimer's disease model. Cell Death Dis. 2018;9(6):655. Published 2018 May 29.

[2]. Hsieh YL, et al. Anti-metastatic activity of MPT0G211, a novel HDAC6 inhibitor, in human breast cancer cells in vitro and in vivo. Biochim Biophys Acta Mol Cell Res. 2019;1866(6):992-1003.

[3]. Tu HJ, et al. The anticancer effects of MPT0G211, a novel HDAC6 inhibitor, combined with chemotherapeutic agents in human acute leukemia cells. Clin Epigenetics. 2018;10(1):162. Published 2018 Dec 29.

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