(-)-Huperzine A

Cat. No.: HY-17387

CAS No.: 102518-79-6

Molecular Formula: $C_{15}H_{18}N_2O$

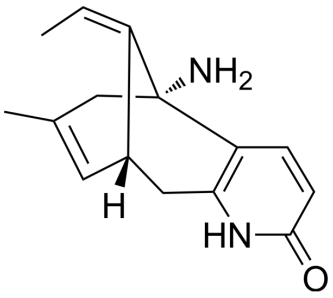
Molecular Weight: 242.32

Target: Cholinesterase (ChE); Apoptosis; iGluR

Pathway: Neuronal Signaling; Apoptosis; Membrane Transporter/Ion Channel

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 (412.68 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.1268 mL	20.6339 mL	41.2677 mL
5 mM	0.8254 mL	4.1268 mL	8.2535 mL
10 mM	0.4127 mL	2.0634 mL	4.1268 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 (10.32 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.32 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

(-)-Huperzine A (Huperzine A) is an alkaloid isolated from Huperzia serrata, with neuroprotective activity. (-)-Huperzine A is a potent, highly specific, reversible and blood-brain barrier penetrant inhibitor of acetylcholinesterase (AChE), with an IC_{50} of 82 nM. (-)-Huperzine A also is non-competitive antagonist of N-methyl-D-aspartate glutamate (NMDA) receptor. (-)-Huperzine A is developed for the research of neurodegenerative diseases, including Alzheimer's disease[1][2][3][4][5].

IC_{50} & Target		
	AChE	NMDA Receptor

In Vitro	(-)-Huperzine A (1 μM; 2 hours) attenuates Aβ23-35 (20 μM)-induced neuronal injury[2]. (-)-Huperzine A (100 μM) reversibly inhibits the NMDA-induced current (IC_{50}=126 μM) in whole-cell voltage-clamp recording in CA1 pyramidal neurons acutely dissociated from rat hippocampus[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	(-)-Huperzine A (0.1-0.2 mg/kg; i.p.; daily; for 12 days) can alleviate the cognitive dysfunction and neuronal degeneration induced by i.c.v. infusion of beta-amyloid protein-(1-40) in rats[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats (220-280 g)[5]
Dosage:	0.1 mg/kg, 0.2 mg/kg
Administration:	Intraperitoneal injection, daily, for 12 days
Result:	Partly reversed the down-regulation of anti-apoptotic Bcl-2 and the up-regulation of pro-apoptotic Bax and P53 proteins and reduced the apoptosis that normally followed b-amyloid injection; alleviated the cognitive dysfunction induced by b-amyloid protein-(1-40).

CUSTOMER VALIDATION

- Cell Metab. 2022 Feb 7;34(3):424-440.e7.
- Aging (Albany NY). 2022 Oct 12;14(19):8077-8094.
- bioRxiv. 2023 Jun 3.

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REFERENCES

[1]. MA Xiao-Chao, XIN Jian, WANG Hai-Xue, et al. Acute effects of huperzine A and tacrine on rat liver. Acta Pharmacol ogica Sinica, 2003, 24(3):247-250.

[2]. Rui Wang, et al. Progress in studies of huperzine A, a natural cholinesterase inhibitor from Chinese herbal medicine. Acta Pharmacol Sin. 2006 Jan;27(1):1-26.

[3]. J M Zhang, et al. Huperzine A, a nootropic alkaloid, inhibits N-methyl-D-aspartate-induced current in rat dissociated hippocampal neurons.Neuroscience. 2001;105(3):663-9

[4]. Maung Kyaw Moe Tun, et al. The pharmacology and therapeutic potential of (−)-huperzine A. J Exp Pharmacol. 2012; 4: 113–123.

[5]. R Wang, et al. Huperzine A attenuates cognitive dysfunction and neuronal degeneration caused by beta-amyloid protein-(1-40) in rat. Eur J Pharmacol. 2001 Jun 15;421(3):149-56.

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