Harmine hydrochloride

Cat. No.: HY-N0737

CAS No.: 343-27-1

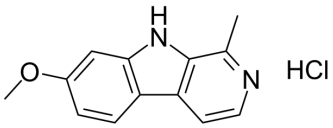
Molecular Formula: $C_{13}H_{13}ClN_2O$

Molecular Weight: 248.71

Target: DYRK; 5-HT Receptor

Pathway: Protein Tyrosine Kinase/RTK; GPCR/G Protein; Neuronal Signaling

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

H_2O : 10 mg/mL (40.21 mM; ultrasonic and warming and heat to 60°C)
DMSO : 5 mg/mL (20.10 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.0207 mL	20.1037 mL	40.2075 mL
5 mM	0.8041 mL	4.0207 mL	8.0415 mL
10 mM	0.4021 mL	2.0104 mL	4.0207 mL

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

BIOLOGICAL ACTIVITY

Description

Harmine Hydrochloride (Telepathine Hydrochloride) is a natural DYRK inhibitor with anticancer and anti-inflammatory activities. Harmine has a high affinity of 5-HT_{2A} serotonin receptor, with an K_i of 397 nM[1].

IC_{50} & Target

5-HT_{2A} Receptor 397 nM (Ki)	DYRK1A

In Vitro

Harmine inhibits tau phosphorylation by DYRK1A by selected DANDYs, with an IC_{50} of 190 nM[2]. Harmine negatively regulates homologous recombination (HR) by interfering Rad51 recruitment, resulting in severe cytotoxicity in hepatoma cells. Furthermore, NHEJ inhibitor Nu7441 markedly sensitizes Hep3B cells to the anti-proliferative effects of Harmine[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

It is shown that brain water content is significantly increased in the TBI group. Treatment with Harmine significantly reduces the tissue water content at 1, 3 and 5 days, compared with the TBI group. Harmine treatment significantly reduces the escape latency at 3 and 5 days, compared with the TBI group. Post-TBI administration of Harmine significantly improves the motor function recovery of the rats at 1, 3 and 5 days following TBI, compared with the TBI group without Harmine treatment. The neuronal survival rate in the Harmine-treated group is significantly increased, compared with the TBI group.

	Administration of Harmine results in marked elevation in the expression of GLT-1, compared with the TBI group. The administration of Harmine significantly reduces the expression of caspase 3, compared with the TBI group[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Stem Cell. 2022 Apr 7;29(4):545-558.e13.
- Sci Adv. 2023 Dec 22;9(51):eadi5683.
- J Biomed Sci. 2022 Jun 2;29(1):34.
- Int Immunopharmacol. 2023 May 5;119:110208.
- Prog Neuropsychopharmacol Biol Psychiatry. 2017 Jun 15;79(Pt B):258-267.

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REFERENCES

[1]. Glennon RA, et al. Binding of beta-carbolines and related agents at serotonin (5-HT(2) and 5-HT(1A)), dopamine (D(2)) and benzodiazepine receptors. Drug Alcohol Depend. 2000 Aug 1;60(2):121-32.

[2]. Neumann F, et al. DYRK1A inhibition and cognitive rescue in a Down syndrome mouse model are induced by new fluoro-DANDY derivatives. Sci Rep. 2018 Feb 12;8(1):2859.

[3]. Zhang L, et al. Harmine suppresses homologous recombination repair and inhibits proliferation of hepatoma cells. Cancer Biol Ther. 2015;16(11):1585-92.

[4]. Zhong Z, et al. Treatment with harmine ameliorates functional impairment and neuronal death following traumatic brain injury. Mol Med Rep. 2015 Dec;12(6):7985-91.

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