Higenamine hydrochloride

Cat. No.: HY-N2037A

CAS No.: 11041-94-4

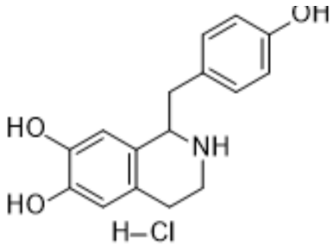
Molecular Formula: $C_{16}H_{18}ClNO_3$

Molecular Weight: 307.77

Target: MDM-2/p53; ROS Kinase; Apoptosis; MAP3K

Pathway: Apoptosis; Protein Tyrosine Kinase/RTK; MAPK/ERK Pathway

Storage: 4°C, sealed storage, away from moisture and light
* In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (324.92 mM; Need ultrasonic)
H_2O : 10 mg/mL (32.49 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.2492 mL	16.2459 mL	32.4918 mL
	5 mM	0.6498 mL	3.2492 mL	6.4984 mL
	10 mM	0.3249 mL	1.6246 mL	3.2492 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (324.92 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (6.76 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (6.76 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Higenamine hydrochloride is a selective LSD1 inhibitor (IC_{50}=1.47 μM) that can be isolated from aconite. Higenamine hydrochloride has anti-inflammatory and antibacterial activity. Higenamine (Norcoclaurine) can attenuate IL-1β-induced Apoptosis through ROS-mediated PI3K/Akt signaling pathway. Higenamine hydrochloride protects brain cells from oxygen deprivation. Higenamine can promote bone formation in osteoporosis through the SMAD2/3 pathway. Higenamine hydrochloride can be used to study cancer, inflammation, cardiorenal syndrome and other diseases[1][2][3][4][5][6].

IC_{50} & Target		
	β adrenergic receptor	ASK1

In Vitro

Higenamine hydrochloride (3-100 μM; 72 h) can inhibit the differentiation of MV4-11 and MOLM-13 cells by inhibiting the activity of LSD1[1].
Higenamine hydrochloride (1-100μM; 8 h) can enhance the activity of HO-1 in C6 cells and protect brain cells from cell hypoxia damage [2].
Higenamine hydrochloride (10-50 μM; 8 h) can inhibit apoptosis in C6 cells[2].
Higenamine hydrochloride (10-40 μM; 24 h) can inhibit the production of IL-1β-induced ROS and activate the ROS-mediated PI3K/Akt signaling pathway, which has anti-apoptotic activity in HNPCs[3].
Higenamine hydrochloride (0.08-250 μM; 0.5-24 h) promotes phosphorylation of SMAD2/3 in a time- and dose-dependent manner in BMSCs[6].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	MV4-11, MOLM-13
Concentration:	3 μM, 10 μM, 30 μM, 100 μM
Incubation Time:	72 h
Result:	Could up-regulate the expression levels of LSD1 substrate H3K4me1 and H3K4me2 in a dose-dependent manner, but did not affect the expression levels of H3K4me3, H3 and LSD1. Promoted P53 expression in a dose-dependent manner.

Western Blot Analysis[2]

Cell Line:	C6
Concentration:	1 μM, 5μM, 10 μM, 50 μM,100 μM
Incubation Time:	8 h
Result:	Increased HO-1 expression in a concentration-dependent manner under hypoxia and normoxia conditions.

Real Time qPCR[1]

Cell Line:	MV4-11, MOLM-13
Concentration:	3 μM, 10 μM, 30 μM, 100 μM
Incubation Time:	72 h
Result:	Significantly down-regulated the expression levels of HoxA9 and Meis1 in leukemia cells in a dose-dependent manner.

In Vivo

Higenamine hydrochloride (10 mg/kg; Intraperitoneal injection; Single dose) can significantly reduce the inflammation and infarct size of cerebral ischemic injury caused by middle cerebral artery occlusion (MCAO) in Sprague-Dawley rats[2].
Higenamine hydrochloride (0.5-4.5 mg/kg; Single dose) improves cardiac and renal function in rats with cardio-renal syndrome (CRS) and alleviates cardiac and renal fibrosis by targeting ASK1/MAPK (ERK, P38)/NF-kB signaling pathway in Sprague-Dawley rats[4].
Higenamine hydrochloride (20 mg/kg-30 mg/kg; Intraperitoneal injection; Once daily for 60 days) promotes bone formation and prevents accelerated bone loss in SAMP6 mice[6].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Spontaneous osteoporosis SAMP6 mice model[6]

Dosage:	10 mg/kg, 20 mg/kg
Administration:	Intraperitoneal injection (i.p.);Once daily for 60 days
Result:	Significantly increased the expression of P1NP and OCN (P1NP and OCN are markers of bone formation).

CUSTOMER VALIDATION

- Nutrients. 2024 May 22.
- J Pharmaceut Biomed. 2020, 113870.

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REFERENCES

[1]. Fang Y, et al. Discovery of higenamine as a potent, selective and cellular active natural LSD1 inhibitor for MLL-rearranged leukemia therapy. Bioorg Chem. 2021 Apr;109:104723.

[2]. Ha YM, et al. Higenamine reduces HMGB1 during hypoxia-induced brain injury by induction of heme oxygenase-1 through PI3K/Akt/Nrf-2 signal pathways. Apoptosis. 2012 May;17(5):463-74.

[3]. Zhu X, et al. Higenamine mitigates interleukin-1β-induced human nucleus pulposus cell apoptosis by ROS-mediated PI3K/Akt signaling. Mol Cell Biochem. 2021 Nov;476(11):3889-3897.

[4]. Deng T, et al. Higenamine Improves Cardiac and Renal Fibrosis in Rats With Cardiorenal Syndrome via ASK1 Signaling Pathway. J Cardiovasc Pharmacol. 2020 Jun;75(6):535-544.

[5]. Erasto, Paul et al. Evaluation of Antimycobacterial Activity of Higenamine Using Galleria mellonella as an In Vivo Infection Model. Natural products and bioprospecting vol. 8,1 (2018): 63-69.

[6]. Dong, Hui et al. Higenamine Promotes Osteogenesis Via IQGAP1/SMAD4 Signaling Pathway and Prevents Age- and Estrogen-Dependent Bone Loss in Mice. Journal of bone and mineral research : the official journal of the American Society for Bone and Mineral Research vol. 38,5 (2023): 775-791.

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