Hydralazine hydrochloride

Cat. No.: HY-B0464

CAS No.: 304-20-1

Molecular Formula: $C_8H_9ClN_4$

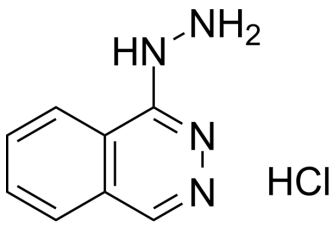
Molecular Weight: 196.64

Target: Bcl-2 Family; Caspase; Apoptosis

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

H_2O : 25 mg/mL (127.14 mM; Need ultrasonic)

DMSO : 25 mg/mL (127.14 mM; ultrasonic and adjust pH to 11 with NaOH)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.0854 mL	25.4272 mL	50.8544 mL
5 mM	1.0171 mL	5.0854 mL	10.1709 mL
10 mM	0.5085 mL	2.5427 mL	5.0854 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 8.33 mg/mL (42.36 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (10.58 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (10.58 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Hydralazine hydrochloride is an antihypertensive agent. Hydralazine hydrochloride can inhibit mitochondrial fission and human peritoneal mesothelial cell proliferation. Hydralazine hydrochloride has immunomodulation and anti-migratory effect. Hydralazine hydrochloride activates the intrinsic pathway of apoptosis and causes DNA damage[1][2][3][4][5].

IC_{50} & Target

Bcl-2	Bcl-xL	Caspase-8	Caspase-9
Caspase 3			

In Vitro

Hydralazine hydrochloride (40-600 μM, 24-48 h) induces caspase-dependent apoptosis and activates mitochondrial apoptotic events in leukemic T cells[1].
Hydralazine hydrochloride (1 μM, 40 min) prevents H_2O_2-induced mitochondrial fragmentation and depolarization in ATCC-CCL2 cells[2].
Hydralazine hydrochloride (3-100 μg/mL, 1-5 days) inhibits human peritoneal mesothelial cell proliferation[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Human peritoneal mesothelial cell (HPMC)
Concentration:	3μg/mL, 6 μg/mL, 12 μg/mL, 25 μg/mL, 50 μg/mL, 100 μg/mL
Incubation Time:	5 days
Result:	Caused a dose-dependent inhibition of HPMC growth.

Apoptosis Analysis[3]

Cell Line:	Jurkat, CEM-6, MOLT-4
Concentration:	40 μM, 80 μM, 200 μM, 400 μM, 600 μM
Incubation Time:	16 h, 24 h, 48 h
Result:	Observed significant apoptosis in Jurkat and MOLT-4 cells at concentrations of 200 μM. Induced a slight decrease in DNMT1 protein expression at 24 hr and nearly a complete loss after 48 hr. Observed proteolytic processing of the initiator caspases-8 and -9, as well as cleavage of the effector caspase-3. Resulted in generation of ROS and disruption of ΔΨm in Jurkat cells. Observed that overexpression of Bcl-2 and Bcl-xL proteins prevented cell death.

In Vivo

Hydralazine hydrochloride (5 mg/kg, Intraperitoneal injection, once a day for 4-6 weeks) plays an immunomodulation role of pro-regeneration in Spinal cord injury mice[4].
Hydralazine hydrochloride (14 mg/kg, supplemented in drinking water for 15 days) reduces leukocyte migration in spontaneously hypertensive and normotensive rats[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Spinal cord injury mice[4]
Dosage:	5 mg/kg
Administration:	Intraperitoneal injection (i.p.)
Result:	Improved not only motor function and hypersensitivity but also spontaneous pain and emotion response in SCI mice. Inhibited both of BMDM recruitment and acrolein accumulation in the lesion epicenter of spinal cord in mice.

Animal Model:	Spontaneously hypertensive rats (SHR)[5]
Dosage:	14 mg/kg
Administration:	Supplemented in drinking water

Result:	Reduced systolic blood pressure values and the number of adherent and migrating leukocytes in SHR. Reduce the number of adherent and migrating leukocytes in SHR. Decreased the levels of ICAM-1 mRNA.

CUSTOMER VALIDATION

- J Pharm Anal. 2023 Aug 11.
- Neurosci Bull. 2020 Oct;36(10):1158-1170.

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REFERENCES

[1]. Fang C C, et al. Hydralazine inhibits human peritoneal mesothelial cell proliferation and collagen synthesis [J]. Nephrology Dialysis Transplantation, 1996, 11(11): 2276-2281.

[2]. Kalkhoran S B, et al. Hydralazine protects the heart against acute ischaemia/reperfusion injury by inhibiting Drp1-mediated mitochondrial fission [J]. Cardiovascular Research, 2022, 118(1): 282-294.

[3]. Ruiz-Magaña M J, et al. The antihypertensive drug hydralazine activates the intrinsic pathway of apoptosis and causes DNA damage in leukemic T cells [J]. Oncotarget, 2016, 7(16): 21875.

[4]. Quan X, et al. Hydralazine plays an immunomodulation role of pro-regeneration in a mouse model of spinal cord injury [J]. Experimental Neurology, 2023, 363: 114367.

[5]. Rodrigues S F, et al. Hydralazine reduces leukocyte migration through different mechanisms in spontaneously hypertensive and normotensive rats [J]. European journal of pharmacology, 2008, 589(1-3): 206-214.

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