Diphenidol hydrochloride

Cat. No.: HY-A0082

CAS No.: 3254-89-5

Molecular Formula: $C_{21}H_{28}ClNO$

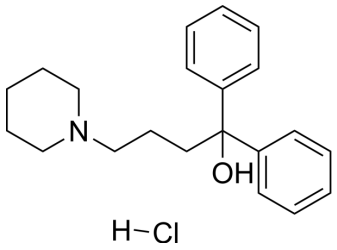
Molecular Weight: 345.91

Target: mAChR; Sodium Channel

Pathway: GPCR/G Protein; Neuronal Signaling; Membrane Transporter/Ion Channel

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

DMSO : ≥ 46 mg/mL (132.98 mM)

H_2O : 16.67 mg/mL (48.19 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8909 mL	14.4546 mL	28.9093 mL
	5 mM	0.5782 mL	2.8909 mL	5.7819 mL
	10 mM	0.2891 mL	1.4455 mL	2.8909 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 (7.23 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.23 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Diphenidol hydrochloride (Difenidol hydrochloride) is a non-selective muscarinic M_1-M_4 receptor antagonist, has anti-arrhythmic activity. Diphenidol hydrochloride is also a potent non-specific blocker of voltage-gated ion channels (Na^+, K^+, and Ca^{2+}) in neuronal cells. Diphenidol hydrochloride can be used in the study of antivertigo and antinausea[1][2][3][4][5].

IC_{50} & Target				
	mAChR3	mAChR2	mAChR1	mAChR4

In Vitro

Diphenidol hydrochloride inhibits sodium currents and produces spinal anesthesia, and at -70 and -100 mV holding potentials, N2A cells IC_{50} were 0.77 and 62.6 μM, respectively [3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Diphenidol hydrochloride (2, 10 μmoL/kg, intraperitoneal injection) is used to reduce neuropathic pain and TNF-α overexpression in rats after chronic systolic injury[4].

Diphenidol hydrochloride (30 mg/kg, injected intravenously) has an inhibitory effect on exercise and morphine-induced vomiting in pica rats[5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Chronic constriction injury (CCI) rat model[4]
Dosage:	2, 10 μmoL/kg
Administration:	i.p., berore surgery, and on postoperative days 3, 6, 7, 11, 13 and 14.
Result:	Increased mechanical withdrawal threshold in a dose-dependent manner and decreased the TNF-α level.

REFERENCES

[1]. Leung YM, et al. Diphenidol inhibited sodium currents and produced spinal anesthesia. Neuropharmacology. 2010 Jun;58(7):1147-52.

[2]. Chen YW, et al. Systemic diphenidol reduces neuropathic allodynia and TNF-alpha overexpression in rats after chronic constriction injury. Neurosci Lett. 2013 Sep 27;552:62-5.

[3]. Takeda N, et al. Neuropharmacological mechanisms of emesis. I. Effects of antiemetic drugs on motion- and apomorphine-induced pica in rats. Methods Find Exp Clin Pharmacol. 1995 Nov;17(9):589-90.

[4]. Leung YM, et al. Inhibition of voltage-gated K+ channels and Ca2+ channels by diphenidol. Pharmacol Rep. 2012;64(3):739-44.

[5]. Leung YM, et al. Diphenidol inhibited sodium currents and produced spinal anesthesia. Neuropharmacology. 2010 Jun;58(7):1147-52.

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