Diphenidol

Cat. No.: HY-A0270

CAS No.: 972-02-1

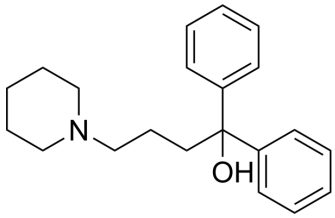
Molecular Formula: $C_{21}H_{27}NO$

Molecular Weight: 309.45

Target: mAChR; Sodium Channel

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

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

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

IC_{50} & Target	mAChR1	mAChR2	mAChR3	mAChR4

In Vitro	Diphenidol 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 (2, 10 μmoL/kg, intraperitoneal injection) is used to reduce neuropathic pain and TNF-α overexpression in rats after chronic systolic injury[4]. Diphenidol (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. Inhibition of voltage-gated K+ channels and Ca2+ channels by diphenidol. Pharmacol Rep. 2012;64(3):739-44.

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

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

[4]. 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.

[5]. 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.

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