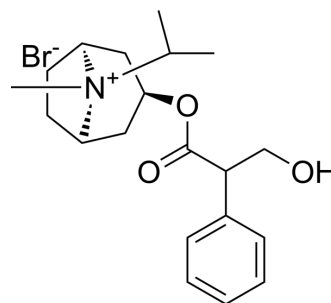


Ipratropium bromide

Cat. No.:	HY-B0241
CAS No.:	22254-24-6
Molecular Formula:	C ₂₀ H ₃₀ BrNO ₃
Molecular Weight:	412.36
Target:	mAChR
Pathway:	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

H₂O : 100 mg/mL (242.51 mM; Need ultrasonic)
 DMSO : ≥ 35 mg/mL (84.88 mM)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	2.4251 mL	12.1253 mL	24.2507 mL
	5 mM	0.4850 mL	2.4251 mL	4.8501 mL
	10 mM	0.2425 mL	1.2125 mL	2.4251 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 50 mg/mL (121.25 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (6.06 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (6.06 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (6.06 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ipratropium bromide (Sch 1000) is a muscarinic receptor antagonist, with IC₅₀s of 2.9 nM, 2 nM, and 1.7 nM for M1, M2, and M3 receptors, respectively. Ipratropium bromide relaxes smooth muscle, can be used in the research for COPD (chronic obstructive pulmonary disease) and asthma^{[1][2][3][4][5]}.

IC₅₀ & Target

mAChR1	mAChR2	mAChR3
--------	--------	--------

In Vitro	<p>Ipratropium bromide (1 nM, 10 nM, 100 nM; 15 min) exerts its toxic effects via disruption of mitochondrial membrane potential^[1].</p> <p>Ipratropium bromide (1 nM-1 μM; 4 h) increases infarct size in isolated perfused heart ischaemia/reperfusion experiments with a dose-responsive manner (EC₅₀=22.7 nM)^[1].</p> <p>Ipratropium bromide (0.001 nM-0.1 mM; 2 h) inhibits adult rat cardiac myocyte growth after 4 h hypoxia treatment^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Viability Assay^[1]</p>								
	<table border="1"> <tr> <td>Cell Line:</td> <td>Adult Rat Cardiac Myocyte</td> </tr> <tr> <td>Concentration:</td> <td>0.001 nM-0.1 mM</td> </tr> <tr> <td>Incubation Time:</td> <td>2 h in dark; prior to 4 h hypoxia</td> </tr> <tr> <td>Result:</td> <td>Resulted cell viability in a dose-dependent manner, with the inhibition rate of 52.7% at 0.1 mM dose.</td> </tr> </table>	Cell Line:	Adult Rat Cardiac Myocyte	Concentration:	0.001 nM-0.1 mM	Incubation Time:	2 h in dark; prior to 4 h hypoxia	Result:	Resulted cell viability in a dose-dependent manner, with the inhibition rate of 52.7% at 0.1 mM dose.
	Cell Line:	Adult Rat Cardiac Myocyte							
	Concentration:	0.001 nM-0.1 mM							
	Incubation Time:	2 h in dark; prior to 4 h hypoxia							
Result:	Resulted cell viability in a dose-dependent manner, with the inhibition rate of 52.7% at 0.1 mM dose.								
In Vivo	<p>Ipratropium bromide (1.0 μg/kg; i.v.; single dose) enhances vagal nerve stimulation inducing bronchoconstriction^[2].</p> <p>Ipratropium bromide (0.04 mg/20 mL and 0.20 mg/20 mL; 30 min, rate=30 mL/30 min) can protect the lungs against the cadmium-induced acute neutrophilic inflammation by reducing the parenchyma inflammatory infiltration of neutrophils^[4].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>								
	<table border="1"> <tr> <td>Animal Model:</td> <td>Guinea-pigs of the Dunkin Hartley strain^[2].</td> </tr> <tr> <td>Dosage:</td> <td>0.1-1 μg/kg</td> </tr> <tr> <td>Administration:</td> <td>Intravenous injection; single dose</td> </tr> <tr> <td>Result:</td> <td>Resulted little blocking effect on post-junctional muscarinic receptors at 0.3 μg/kg, and inhibited ACh-induced bronchoconstriction at 0.5 μg/kg.</td> </tr> </table>	Animal Model:	Guinea-pigs of the Dunkin Hartley strain ^[2] .	Dosage:	0.1-1 μg/kg	Administration:	Intravenous injection; single dose	Result:	Resulted little blocking effect on post-junctional muscarinic receptors at 0.3 μg/kg, and inhibited ACh-induced bronchoconstriction at 0.5 μg/kg.
	Animal Model:	Guinea-pigs of the Dunkin Hartley strain ^[2] .							
	Dosage:	0.1-1 μg/kg							
	Administration:	Intravenous injection; single dose							
	Result:	Resulted little blocking effect on post-junctional muscarinic receptors at 0.3 μg/kg, and inhibited ACh-induced bronchoconstriction at 0.5 μg/kg.							

REFERENCES

- [1]. Fryer AD, et al. MacLagan, Ipratropium bromide potentiates bronchoconstriction induced by vagal nerve stimulation in the guinea-pig. *Eur J Pharmacol*, 1987. 139(2): p. 187-91.
- [2]. Harvey, et al. Maddock, Ipratropium Bromide-Mediated Myocardial Injury in In Vitro Models of Myocardial Ischaemia/Reperfusion. *Toxicol Sci*, 2014.
- [3]. Maria Prat, et al. Discovery of novel quaternary ammonium derivatives of (3R)-quinuclidinyl amides as potent and long acting muscarinic antagonists. *Bioorg Med Chem Lett*. 2015 Apr 15;25(8):1736-1741.
- [4]. Wenhui Zhang, et al. Anti-inflammatory effects of formoterol and ipratropium bromide against acute cadmium-induced pulmonary inflammation in rats. *Eur J Pharmacol*. 2010 Feb 25;628(1-3):171-8.

Caution: Product has not been fully validated for medical applications. For research use only.

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