Hexamethonium Bromide

Cat. No.: HY-B0569

CAS No.: 55-97-0

Molecular Formula: $C_{12}H_{30}Br_2N_2$

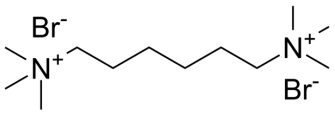
Molecular Weight: 362.19

Target: nAChR; Apoptosis

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; 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 : ≥ 100 mg/mL (276.10 mM)

DMSO : 16.67 mg/mL (46.03 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.7610 mL	13.8049 mL	27.6098 mL
5 mM	0.5522 mL	2.7610 mL	5.5220 mL
10 mM	0.2761 mL	1.3805 mL	2.7610 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 (276.10 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.67 mg/mL (4.61 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.67 mg/mL (4.61 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Hexamethonium Bromide is a non-selective ganglionic nicotinic-receptor antagonist (nAChR) antagonist, with mixed competitive and noncompetitive activity. Hexamethonium Bromide has anti-hypertensive activity. Hexamethonium Bromide attenuates sympathetic activity and blood pressure in spontaneously hypertensive animal models[1][2][3][4].

IC_{50} & Target

nAChR[1]

In Vitro

Hexamethonium Bromide (100 μM; 60 minutes) abolishes epibatidine-induced depolarisations of smooth muscle cells[1].

	The α3β4α5 receptors have greater sensitivity to Hexamethonium Bromide than the other potential ganglionic models, α3β4, α3β2, and α3β2α5[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Hexamethonium Bromide (0.2-25 mg/kg; i.v.) significantly reduces the renal sympathetic nerve activity (RSNA), mean arterial pressure (MAP) and heart rate (HR) in the Wistar rats and the spontaneously hypertensive rats (SHRs)[4]. Hexamethonium Bromide (0.2-1.0 mg/kg; i.v.) treatments show no significant differences in the RSNA, MAP or HR between Wistar rats and SHRs[4]. Hexamethonium Bromide (5.0-25 mg/kg; i.v.) results in a greater reduction in the RSNA and MAP in SHRs compared with Wistar rats[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male normotensive Wistar rats (280-320 g), SHRs[4]
Dosage:	0.2 mg/kg, 1.0 mg/kg, 5.0 mg/kg, 25 mg/kg
Administration:	Intravenous injection
Result:	Significantly reduced the RSNA, MAP and HR in the Wistar rats and the SHRs.

CUSTOMER VALIDATION

- Atherosclerosis. 2019 Feb 22;284:1-10.
- Evid-Based Compl Alt. Dec 12.

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REFERENCES

[1]. Damian J. Williams, et al. Mechanisms involved in nicotinic acetylcholine receptor-induced neurotransmitter release from sympathetic nerve terminals in the mouse vas deferens. PLoS One. 2011; 6(12): e29209.

[2]. Roger L. Papke, et al. Activation and Inhibition of Mouse Muscle and Neuronal Nicotinic Acetylcholine Receptors Expressed in Xenopus Oocytes. J Pharmacol Exp Ther. 2010 May; 333(2): 501–518.

[3]. Brian T Hawkins, et al. Modulation of cerebral microvascular permeability by endothelial nicotinic acetylcholine receptors. Am J Physiol Heart Circ Physiol. 2005 Jul;289(1):H212-9.

[4]. Peng Li, et al. Hexamethonium attenuates sympathetic activity and blood pressure in spontaneously hypertensive rats. Mol Med Rep. 2015 Nov;12(5):7116-22.

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