Vecuronium bromide

Cat. No.: HY-B0118A

CAS No.: 50700-72-6

Molecular Formula: $C_{34}H_{57}BrN_2O_4$

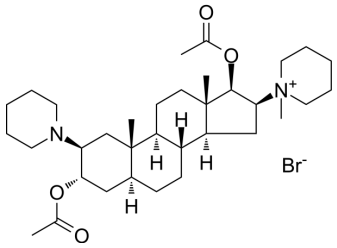
Molecular Weight: 637.73

Target: nAChR

Pathway: Membrane Transporter/Ion Channel; 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

H_2O : ≥ 100 mg/mL (156.81 mM)

DMSO : ≥ 46 mg/mL (72.13 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Concentration / Solvent / Mass	1 mg	5 mg	10 mg
	1 mM	1.5681 mL	7.8403 mL	15.6806 mL
	5 mM	0.3136 mL	1.5681 mL	3.1361 mL
	10 mM	0.1568 mL	0.7840 mL	1.5681 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: ≥ 2.08 mg/mL (3.26 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.26 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (3.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Vecuronium (ORG NC 45) bromide is a non-depolarizing neuromuscular blocking agent that also acts as a nicotinic acetylcholine receptor (nAChR) inhibitor, a muscle relaxant, and can be used for pre-surgical anesthesia[1][2].

In Vitro

Vecuronium bromide (0-100 μM, 15 min) inhibits [^{3}H] norepinephrine (NE) uptake to 65% at 100 μM in adrenal medullary cells[1].

Vecuronium bromide (0-15 μM,72 hours) inhibits cancer cell proliferation and migration in a concentration-dependent manner[2].

Vecuronium bromide (0-15 μM,72 hours) can significantly reduce cell viability by combining with cisplatin[2].

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

Cell Proliferation Assay[2]

Cell Line:	Lung cancer cell line (A549)
Concentration:	0-15 μM
Incubation Time:	72 hours
Result:	Inhibited cell proliferation at concentrations ranging from 5.0 μM to 15 μM.

Cell Cytotoxicity Assay[2]

Cell Line:	Lung cancer cell line (A549)
Concentration:	0-15 μM
Incubation Time:	72 hours
Result:	Resulted in a decrease in cell viability from 10 μM to 15 Mm by combining with cisplatin.

In Vivo

Vecuronium bromide (Intravenous injection, 0-5 μM, every 30 min, 2 hours) attenuates the response of carotid sinus nerve activity (CSNA) to hypoxia in a dose-dependent manner and inhibits the neural response of the carotid body to acetylcholine (ACh) in Wister rats[3].

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

Animal Model:	Wister rats weighing 250-350 g[3]
Dosage:	0-5 μM
Administration:	Intravenous injection; every 30 min; 2 hours
Result:	Significantly diminished CSNA response to hypoxia at the concentration of 5 μM and reduced carotid sinus nerve response to ACh at 0.5 μM.

REFERENCES

[1]. K Uryu, et al. Inhibition by neuromuscular blocking drugs of norepinephrine transporter in cultured bovine adrenal medullary cells. Anesth Analg. 2000 Sep;91(3):546-51.

[2]. Iddrisu BabaYabasin, et al. Anticancer effects of vecuronium bromide and cisatracurium besylate on lung cancer cells (A549), in vitro. Biomedicine & Aging Pathology,Volume 4, Issue 4,

[3]. Ayuko Igarashi,et al. Vecuronium directly inhibits hypoxic neurotransmission of the rat carotid body. Anesth Analg. 2002 Jan;94(1):117-22, table of contents.

[4]. Meretoja, O.A., Vecuronium infusion requirements in pediatric patients during fentanyl-N2O-O2 anesthesia. Anesth Analg, 1989. 68(1): p. 20-4.

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[6]. Shingu, K., et al., [Neuromuscular blocking effects of Org 9426 (rocuronium bromide); a comparative study with vecuronium bromide in Japanese patients]. Masui, 2006. 55(9): p. 1140-8.

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

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