Ropivacaine hydrochloride

Cat. No.: HY-B0563B

CAS No.: 98717-15-8

Molecular Formula: $C_{17}H_{27}ClN_2O$

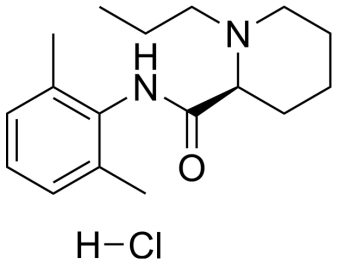
Molecular Weight: 310.86

Target: Potassium Channel; Sodium Channel

Pathway: 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 : 10 mg/mL (32.17 mM; Need ultrasonic)

H_2O : 10 mg/mL (32.17 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.2169 mL	16.0844 mL	32.1688 mL
5 mM	0.6434 mL	3.2169 mL	6.4338 mL
10 mM	0.3217 mL	1.6084 mL	3.2169 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: ≥ 1 mg/mL (3.22 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1 mg/mL (3.22 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1 mg/mL (3.22 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ropivacaine hydrochloride is a potent sodium channel blocker and blocks impulse conduction via reversible inhibition of sodium ion influx in nerve fibrese[1][2]. Ropivacaine is also an inhibitor of K_{2P} (two-pore domain potassium channel) TREK-1 with an IC_{50} of 402.7 μM in COS-7 cell's membrane[3]. Ropivacaine is widely used for neuropathic pain management in vivo[1] .

IC_{50} & Target

IC50: sodium ion influx[1]

IC50: 402.7 μM (TREK-1 in COS-7 cell's membrane)[3]

In Vivo

Epidural administration of Ropivacaine hydrochloride effectively blocks neuropathic pain (both mechanical allodynia and heat hyperalgesia) without induction of analgesic tolerance and significantly delays the development of neuropathic pain produced by peripheral nerve injury[1].

Ropivacaine hydrochloride inhibits pressure-induced increases in filtration coefficient (Kf) without affecting pulmonary artery pressure (Ppa), pulmonary capillary pressures (Ppc), and zonal characteristics (ZC)[2].

Ropivacaine hydrochloride prevents pressure-induced lung edema and associated hyperpermeability as evidence by maintaining PaO2, lung wet-to-dry ratio and plasma volume in levels similar to sham rats[2].

Ropivacaine hydrochloride inhibits pressure-induced NO production as evidenced by decreased lung nitro-tyrosine content when compared to hypertensive lungs[2].

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

Animal Model:	Adult Sprague-Dawley rats (300–400g)[2]
Dosage:	1 μM
Administration:	Infusion (added to the perfusate reservoir)
Result:	Attenuated pressure-dependent increases in filtration coefficient (K_f).

CUSTOMER VALIDATION

- J Exp Clin Cancer Res. 2024 Mar 25;43(1):90.
- Stem Cell Res Ther. 2021 Feb 4;12(1):107.
- Eur Spine J. 2022 Sep 24.
- J Toxicol Sci. 2023;48(3):139-148.

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REFERENCES

[1]. Li TF, et al. Epidural sustained release ropivacaine prolongs anti-allodynia and anti-hyperalgesia in developing and established neuropathic pain. PLoS One. 2015 Jan 24;10(1):e0117321.

[2]. Milan Patel, et al. Ropivacaine Inhibits Pressure-Induced Lung Endothelial Hyperpermeability in Models of Acute Hypertension. Life Sci. 2019 Apr 1;222:22-28.

[3]. Dene Simpson, et al. Ropivacaine: a review of its use in regional anaesthesia and acute pain management. Drugs. 2005;65(18):2675-717.

[4]. Hye Won Shin, et al. The inhibitory effects of bupivacaine, levobupivacaine, and ropivacaine on K2P (two-pore domain potassium) channel TREK-1. J Anesth. 2014 Feb;28(1):81-6.

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