Ropivacaine

Cat. No.: HY-B0563

CAS No.: 84057-95-4

Molecular Formula: $C_{17}H_{26}N_2O$

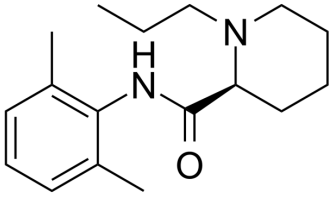
Molecular Weight: 274.4

Target: Potassium Channel; Sodium Channel

Pathway: Membrane Transporter/Ion Channel

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 12.5 mg/mL (45.55 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.6443 mL	18.2216 mL	36.4431 mL
	5 mM	0.7289 mL	3.6443 mL	7.2886 mL
	10 mM	0.3644 mL	1.8222 mL	3.6443 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: ≥ 2.5 mg/mL (9.11 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (9.11 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ropivacain is a potent sodium channel blocker. Ropivacain 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 used for the research of neuropathic pain management[1].

IC_{50} & Target

IC50: sodium ion influx[1]
IC50: 402.7 μM (TREK-1 in COS-7 cell's membrane)[2]

In Vivo

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

Ropivacaine 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 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)[1]
Dosage:	1 μM
Administration:	Infusion (added to the perfusate reservoir)
Result:	Attenuated pressure-dependent increases in filtration coefficient (K_f).

CUSTOMER VALIDATION

- 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

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