Clamikalant sodium

Cat. No.: HY-15208

CAS No.: 261717-22-0

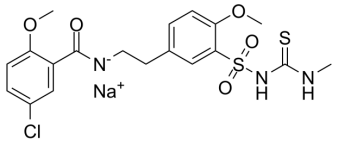
Molecular Formula: $C_{19}H_{21}ClN_3NaO_5S_2$

Molecular Weight: 493.96

Target: Potassium 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 : 50 mg/mL (101.22 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0245 mL	10.1223 mL	20.2446 mL
	5 mM	0.4049 mL	2.0245 mL	4.0489 mL
	10 mM	0.2024 mL	1.0122 mL	2.0245 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 (5.06 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.06 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.06 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Clamikalant sodium (HMR 1098) is an ATP-sensitive potassium (K_{ATP}) channel blocker. Clamikalant sodium can be used for the research of arrhythmia[1].

In Vitro

Clamikalant sodium (HMR 1098; 40 μM) prevents improvement effect of Levosimendam on left ventricular developed pressure (LVDP) recovery rate, abolishes the inhibitory effect of Levosimendan on hypothermic preservation-induced activation of calpain, cleavage of Bid, and apoptosis[2].

Clamikalant sodium (HMR 1098; 30 μM, 24 hours) reduces cellular viability, increases the apoptosis of Neonatal rat cardiomyocytes (NRCs) [3].

Clamikalant sodium (30 μM) decreases the Bcl-2 protein level and increases the Bax protein level in the LPS-exposed NRCs[3]

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

Cell Viability Assay[3]

Cell Line:	Neonatal rat cardiomyocytes (NRCs)
Concentration:	30 μM
Incubation Time:	24 hours
Result:	Reduced cellular viability to 42.8±6.3% compared with the LPS group.

Western Blot Analysis[3]

Cell Line:	Neonatal rat cardiomyocytes (NRCs)
Concentration:	30 μM
Incubation Time:	24 hours
Result:	Decreased the Bcl-2 protein level and increased the Bax protein level.

In Vivo

Clamikalant sodium (HMR 1098; 6.0 mg/kg; 5 min prior to EET administration) completely abolishes the cardioprotection produced by epoxyeicosatrienoic acid (EET)[1].

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

REFERENCES

[1]. Garrett J Gross, et al. Roles of endothelial nitric oxide synthase (eNOS) and mitochondrial permeability transition pore (MPTP) in epoxyeicosatrienoic acid (EET)-induced cardioprotection against infarction in intact rat hearts. J Mol Cell Cardiol. 2013 Ju

[2]. Hai-yan Zhou, et al. Improved myocardial function with supplement of levosimendan to Celsior solution. J Cardiovasc Pharmacol. 2014 Sep;64(3):256-65.

[3]. Xiaohui Zhang, et al. Sarcolemmal ATP-sensitive potassium channel protects cardiac myocytes against lipopolysaccharide-induced apoptosis. Int J Mol Med. 2016 Sep;38(3):758-66.

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