Diltiazem hydrochloride

Cat. No.: HY-14656

CAS No.: 33286-22-5

Molecular Formula: $C_{22}H_{27}ClN_2O_4S$

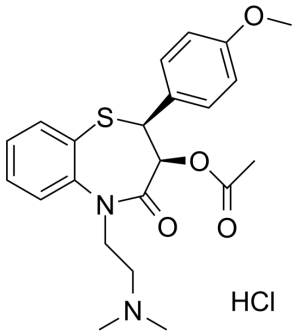
Molecular Weight: 450.98

Target: Calcium Channel

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 : 33.33 mg/mL (73.91 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2174 mL	11.0870 mL	22.1739 mL
	5 mM	0.4435 mL	2.2174 mL	4.4348 mL
	10 mM	0.2217 mL	1.1087 mL	2.2174 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 (221.74 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Diltiazem hydrochloride is a Ca^{2+} influx inhibitor (slow channel blocker or calcium antagonist).

IC_{50} & Target

L-type calcium channel

In Vitro

Benzothiazepine Ca^{2+} antagonist diltiazem hydrochloride interacts with transmembrane segments IIIS6 and IVS6 in the α1 subunit of L-type Ca^{2+} channels[1]. Diltiazem causes a dose-dependent inhibiton of contractions as well as Ca^{2+} influx stimulated by alpha adrenoceptor activation and high-K^+ depolarization. Diltiazem is roughly equally potent in inhibiting contractions induced by high-K^+ and a low concentration of norepinephrine (NE)[2]. Diltiazem also inhibits the Na-dependent Ca-efflux from heart mitochondria. Both the (+)-optical isomers of the cis- and trans-forms of diltiazem inhibit Na-Ca exchange activity with comparable potency (IC_{50} of 10-20 μM)[3].

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

In Vivo

Diltiazem produces a noncompetitive inhibition of Ca^{2+}-induced contractions of depolarized rabbit aorta. Furthermore, there is a lack of parallelism between the smooth muscle effects of removal of [Ca^{2+}]ex and of addition of diltiazem[2].

Diltiazem improves the cardiac microcirculation and function in an experimental model of hyperthyroidism in rats. The treatment of hyperthyroid rats with losartan diltiazem (4.7±0.7%; P < 0.001) significantly reduces the percentage of fibrosis areas in the left ventricle [4]. In conscious spontaneously hypertensive rats (SHR), diltiazem dose-dependently decreases the blood pressure and increases the heart rate after intravenous administration (0.03--1 mg/kg). Oral administration of diltiazem (100 mg/kg) also reduces the blood pressure of SHR[5].

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

CUSTOMER VALIDATION

- Virology. 2020 Jan 2;539:38-48.
- Virology. 2020 Jan 2;539:38-48.
- J Cardiovasc Transl Res. 2023 Jan 30.
- Pharmacol Res Perspect. 2021 Oct;9(5):e00879.
- Pharmacol Res Perspect. 2020 Apr;8(2):e00575.

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REFERENCES

[1]. Kraus RL, et al. Molecular mechanism of diltiazem interaction with L-type Ca2+ channels. J Biol Chem. 1998 Oct 16;273(42):27205-12.

[2]. van Breemen C, et al. The mechanism of inhibitory action of diltiazem on vascular smooth muscle contractility. J Pharmacol Exp Ther. 1981 Aug;218(2):459-63.

[3]. Chiesi M, et al. Stereospecific action of diltiazem on the mitochondrial Na-Ca exchange system and on sarcolemmal Ca-channels. Biochem Pharmacol. 1987 Sep 1;36(17):2735-40.

[4]. Freitas F, et al. Cardiac microvascular rarefaction in hyperthyroid rats is reversed by losartan, diltiazem, and propranolol. Fundam Clin Pharmacol. 2015 Feb;29(1):31-40.

[5]. Sato M, et al. Hypotensive effects of diltiazem hydrochloride in the normotensive, spontaneously hypertensive and renal hypertensive rats (author's transl). Nihon Yakurigaku Zasshi. 1979 Mar;75(2):99-106.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA