Diltiazem

Cat. No.: HY-B0632

CAS No.: 42399-41-7

Molecular Formula: $C_{22}H_{26}N_2O_4S$

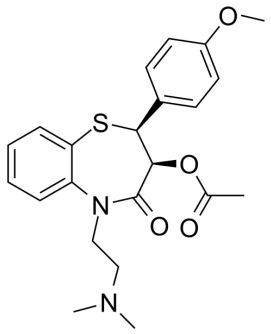
Molecular Weight: 414.52

Target: Calcium Channel

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (120.62 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4124 mL	12.0621 mL	24.1243 mL
5 mM	0.4825 mL	2.4124 mL	4.8249 mL
10 mM	0.2412 mL	1.2062 mL	2.4124 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.03 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Diltiazem is an orally active L-type Ca^{2+} channel blocker. Diltiazem shows antihypertensive and antiarrhythmic effects. Diltiazem can be used for the research of cardiac arrhythmia, hypertension, and angina pectoris[1][2][3].

IC_{50} & Target

L-type calcium channel

In Vitro

Diltiazem (200 μM) elicits a use-dependent blockade that proceeded within a relatively small number of pulses[1].
Diltiazem reduces Ca^{2+} influx by accelerating inactivation during action potentials, and that the use-dependent blockade is due to increases in the number of channels in a sustained closed state[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Diltiazem (100 mg/kg; p.o.; for 4 weeks) prevents aortic aneurysm formation in a blood pressure-independent manner[3].
Diltiazem limits aortic aneurysm formation in mice by a blood pressure-independent anti-inflammatory effect on monocytic

cells[3].

Diltiazem (2 mg/kg; i.v.) exhibits $T_{1/2}$ of 61.2 min, CL_{el} of 3.2 mL/min in rats[4].

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

Animal Model:	Male ApoE$^{-/-}$ mice, angiotensin II induced aneurysms[3]
Dosage:	100 mg/kg
Administration:	Oral administration, in drinking water, for 4 weeks
Result:	Srongly reduced the vascular remodeling but also lowered the blood pressure.

Animal Model:	Rat (200-250 g)[4]
Dosage:	2 mg/kg (Pharmacokinetic Analysis)
Administration:	Intravenous injection
Result:	$T_{1/2}$ (61.2 min), CL_{el} (3.2 mL/min)

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]. Yoshinari Niimi, et al. Diltiazem facilitates inactivation of single L-type calcium channels in guinea pig ventricular myocytes. Jpn Heart J. 2003 Nov;44(6):1005-14.

[2]. S Lin Tang, et l. Structural Basis for Diltiazem Block of a Voltage-Gated Ca2+ Channel. Mol Pharmacol. 2019 Oct; 96(4): 485-492.

[3]. Anja Mieth , et al. L-type calcium channel inhibitor diltiazem prevents aneurysm formation by blood pressure-independent anti-inflammatory effects. Hypertension. 2013 Dec;62(6):1098-104.

[4]. S. J. Downing, et al. Diltiazem pharmacokinetics in the rat and relationship between its serum concentration and uterine and cardiovascular effects. Br J Pharmacol. 1987 Aug; 91(4): 735-745.

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