Cilnidipine

Cat. No.: HY-17404

CAS No.: 132203-70-4

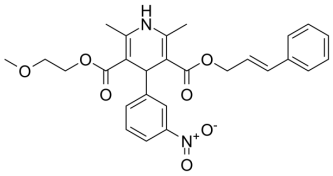
Molecular Formula: $C_{27}H_{28}N_2O_7$

Molecular Weight: 492.52

Target: Calcium Channel

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (203.04 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0304 mL	10.1519 mL	20.3037 mL
5 mM	0.4061 mL	2.0304 mL	4.0607 mL
10 mM	0.2030 mL	1.0152 mL	2.0304 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 (5.08 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.08 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cilnidipine is a long-acting, second-generation dihydropyridine Ca^{2+}-channel blocker on L and N-type Ca^{2+} channel[1][2][3][4]. Antihypertensive effects[5].

In Vitro

Cilnidipine inhibits the L-type current with an IC_{50} of 100 nM in neurons pretreated with omegaCgTx plus omegaAgTx[1].
The IC_{50} for Cilnidipine in respect of the N-type current is 200 nM[1].
Cilnidipine dose- and time-dependently inhibits Ba^{2+} currents in A7r5 cells with the IC_{50} at 10 nM after 10 min[2].
Cilnidipine dose-dependently inhibits depolarization- and Ca^{2+}-induced contractions of rat aortic rings, with an IC_{50} of 10 nM at 10 min[2].
The viability of nPC12 cells show no significant change up to 150 μM Cilnidipine, but it decreases slightly in the cells treated with greater than 200 μM Cilnidipine[3].

Cilnidipine (100 μM, 2 hours) treatment increases the expression of p85aPI3K p-Akt, p-GSK-3β, and heat shock transcription factor (HSTF-1), and decreases levels of cytosolic cytochrome c, activated caspase 3, and cleaved PARP[3].

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

Cell Viability Assay

Cell Line:	Neuronally differentiated PC12 (nPC12) cells
Concentration:	0, 1, 5, 10, 25, 50, 100, 150, and 200 μM
Incubation Time:	Treated for 2 hours; cell viability was measured after 24 hours
Result:	Cell viability was not affected by low concentrations up to 150 μM, but it was slightly decreased at 200 μM.

Western Blot Analysis

Cell Line:	nPC12 cells
Concentration:	100 μM
Incubation Time:	2 hours
Result:	Increased the IRs of p58a PI3K, p-Akt, p-GSK-3β, and HSTF-1 and decreased the Immunoreactivities (IRs) of cytosolic cytochrome c, activated caspase 3 (17 kDa), and cleaved PARP (85 kDa).

In Vivo

Cilnidipine has potent inhibitory actions on N-type as well as L-type voltage-dependent Ca^{2+}-channel in rat dorsal root ganglion neurons[1].

Administration of Cilnidipine (10 mg/kg) and Nimodipine (10 mg/kg) significantly attenuates the immobilized stress-induced behavioral changes and restored memory deficits along with normalization of the corticosterone levels[4].

Cilnidipine and Nimodipine produce comparable beneficial effects in restoring immobilization stress subjected mice[4].

Oral administration of Cilnidipine (3 mg/kg) markedly lowers both systolic and diastolic blood pressure 1 hr after administration in 2K1C renal hypertensive dogs[5].

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

Animal Model:	Swiss albino mice weighing 25±5 g[4]
Dosage:	5 and 10 mg/kg
Administration:	administered i.p. 30 min prior to immobilization stress
Result:	Cilnidipine (10 mg/kg, i.p.) and nimodipine (10 mg/kg, i.p.) 30 min prior to subjecting immobilization stress resulted in significant attenuation of immobilization stress-induced decrease in locomotor activity. Administration with Cilnidipine (5 mg/kg, i.p.) and Nimodipine (5 mg/kg, i.p.) did not show any significant effect on the stressed mice. Administration of Cilnidipine (10 mg/kg, i.p.) and Nimodipine (10 mg/kg, i.p.) in the non-stressed mice, and vehicle in the stressed mice did not modulate locomotor activity in a significant manner.

REFERENCES

[1]. S Fujii,et al. Effect of cilnidipine, a novel dihydropyridine Ca^{2+}-channel antagonist, on N-type Ca^{2+} channel in rat dorsal root ganglion neurons. J Pharmacol Exp Ther. 1997 Mar;280(3):1184-91.

[2]. Matthias Löhn, et al. Cilnidipine is a novel slow-acting blocker of vascular L-type calcium channels that does not target protein kinase C. J Hypertens. 2002 May;20(5):885-93.

[3]. Young Joo Lee, et al. Cilnidipine mediates a neuroprotective effect by scavenging free radicals and activating the phosphatidylinositol 3-kinase pathway. J Neurochem. 2009 Oct;111(1):90-100.

[4]. Naresh Kumar, et al. Anti-stress effects of cilnidipine and nimodipine in immobilization subjected mice. Physiol Behav. 2012 Mar 20;105(5):1148-55.

[5]. A Takahara, et al. [Antihypertensive effects of repeated oral administration of cilnidipine, a novel calcium antagonist, in 2K1C renal hypertensive dogs]. Nihon Yakurigaku Zasshi. 1995 Oct;106(4):279-87.

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