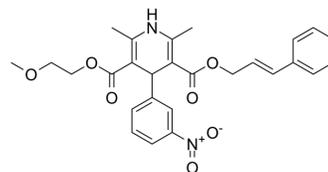


Cilnidipine

Cat. No.:	HY-17404
CAS No.:	132203-70-4
Molecular Formula:	C ₂₇ H ₂₈ N ₂ O 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 Concentration	Mass		
		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

- 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
- 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 Ca²⁺-channel blocker on L and N-type Ca²⁺ channel^{[1][2][3][4]}. Antihypertensive effects^[5].

In Vitro

Cilnidipine inhibits the L-type current with an IC₅₀ of 100 nM in neurons pretreated with omegaCgTx plus omegaAgTx^[1]. The IC₅₀ for Cilnidipine in respect of the N-type current is 200 nM^[1].
Cilnidipine dose- and time-dependently inhibits Ba²⁺ currents in A7r5 cells with the IC₅₀ at 10 nM after 10 min^[2].
Cilnidipine dose-dependently inhibits depolarization- and Ca²⁺-induced contractions of rat aortic rings, with an IC₅₀ 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-3 β , 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-3 β , 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 Ca²⁺-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 \pm 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 Ca²⁺-channel antagonist, on N-type Ca²⁺ 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.

Caution: Product has not been fully validated for medical applications. For research use only.

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