Nilvadipine

Cat. No.: HY-14284

CAS No.: 75530-68-6

Molecular Formula: $C_{19}H_{19}N_3O_6$

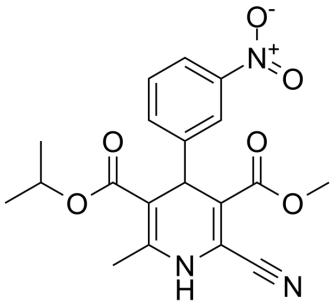
Molecular Weight: 385.37

Target: Calcium Channel

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (129.75 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5949 mL	12.9745 mL	25.9491 mL
5 mM	0.5190 mL	2.5949 mL	5.1898 mL
10 mM	0.2595 mL	1.2975 mL	2.5949 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: ≥ 3.33 mg/mL (8.64 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 3.33 mg/mL (8.64 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3.33 mg/mL (8.64 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Nilvadipine is a potent calcium channel antagonist, and the IC_{50} value is around 0.1 nM.

IC_{50} & Target

IC50: 0.1 nM (Calcium channel)[1]

In Vitro

In an in vitro experiment on inhibition of migration of rat aortic smooth muscle cells, using Zymosan-activated air pouch exudate as a chemoattractant in modified Boyden chambers.The IC_{50} value is 0.033 nM for Nilvadipine (FR34235). Effects of

	Nilvadipine on proliferation of rat aortic smooth muscle cells and rabbit platelet aggregation is also examined. Nilvadipine should be useful for preventing and treating atherosclerosis. Inhibition of smooth muscle cell migration is thought to be its mechanism of antiatherogenic activity[2]. The antioxidant effect of calcium antagonist Nilvadipine is studied by means of rat myocardial membrane lipid peroxidation with a nonenzymatic active oxygen-generating system (DHF/FeC13-ADP) with IC_{50} of 25.1 μM. Nilvadipine shows antioxidant effects both before and after the addition of active oxygen, and reduces the dihydroxyfumarate (DHF) auto-oxidation rate, is chain-breaking and preventive antioxidants. Nicardipine, which shows an antioxidant effect only before exposure to active oxygen and reduced the DHF auto-oxidation rate, is mainly a preventive antioxidant[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	The antiatherogenic activity of Nilvadipine (FR34235), a calcium antagonist, is examined in rabbits with carotid arteries sheathed with polyethylene cuffs, and compared with that of Nifedipine, Verapamil and Diltiazem. Nilvadipine is given intramuscularly in daily doses of 0.01-10 mg/kg for 3 weeks, starting on the day of cuff-placement. FR34235 dose-dependently inhibits the cuff-induced intimal thickening[2]. Nilvadipine affords significant protection against thinning of retinal layers in the RCS rat during retinal degeneration. Electron microscopy shows that marked irregularity in the photoreceptor OS in the untreated retina[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [4]	Rat[4] In the present study, 3- to 5-week-old inbred RCS ($rdy^{-/-}$) rats reared in cyclic light conditions (12 hours on-12 hours off) are used. Nilvadipine and Nifedipine are dissolved in a mixture of ethanol, polyethylene glycol 400, and distilled water (2:1:7) at a concentration of 0.1 mg/mL, diluted twice with physiological saline before use, and injected intraperitoneally (1.0 mL/kg) into anesthetized rats every day early in the morning for 2 weeks. In control rats, the same solution without Nilvadipine or Nifedipine (vehicle solution) is administered similarly. Nicardipine and Diltiazem are dissolved in PBS at 0.25 mg/mL and 1 mg/mL, respectively, and injected intraperitoneally (1 mL/kg), similarly to the other agonists. As a control, the same volume of a mixture of ethanol, polyethylene glycol 400, and distilled water (2:1:7) or PBS is administered. Before administration, the pH of all drug solutions is adjusted to approximately 7.4. The concentrations of these drugs administered to RCS rats are determined by their concentrations in oral administration to human patients with hypertension for 1 day in our clinical practice (Nilvadipine, 0.05-0.3 mg/kg; Nifedipine, 0.1-0.5 mg/kg; Nicardipine, 0.2-1 mg/kg; and Diltiazem, 0.3-3 mg/kg). MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Nomoto A, et al. Smooth muscle cell migration induced by inflammatory cell products and its inhibition by a potent calcium antagonist, Nilvadipine. Atherosclerosis. 1988 Aug;72(2-3):213-9.

[2]. Nomoto A, et al. Antiatherogenic activity of FR34235 (Nilvadipine), a new potent calcium antagonist. Effect on cuff-induced intimal thickening of rabbit carotid artery. Atherosclerosis. 1987 Apr;64(2-3):255-61.

[3]. Sugawara H, et al. Antioxidant effects of calcium antagonists on rat myocardial membrane lipid peroxidation. Hypertens Res. 1996 Dec;19(4):223-8.

[4]. Yamazaki H, et al. Preservation of retinal morphology and functions in royal college surgeons rat by Nilvadipine, a Ca(2+) antagonist. Invest Ophthalmol Vis Sci. 2002 Apr;43(4):919-26.

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

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