Lacidipine

Cat. No.: HY-B0347

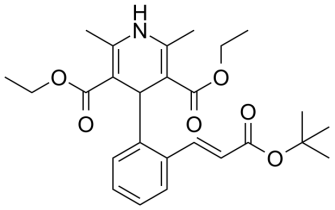
CAS No.: 103890-78-4

Molecular Formula: $C_{26}H_{33}NO_6$

Molecular Weight: 455.54

Target: Calcium Channel; Reactive Oxygen Species; Caspase; Apoptosis

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Apoptosis



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 (109.76 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1952 mL	10.9760 mL	21.9520 mL
5 mM	0.4390 mL	2.1952 mL	4.3904 mL
10 mM	0.2195 mL	1.0976 mL	2.1952 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 (5.49 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.49 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Lacidipine is an orally active and highly selective L-type calcium channel blocker that acts on smooth muscle calcium channels, primarily dilates peripheral arteries, reduces peripheral resistance, and has long-lasting anti-hypertensive activity. Lacidipine protects HKCs from apoptosis induced by ATP depletion and recovery by modulating the caspase-3 pathway. Lacidipine can be used in studies of hypertension, atherosclerosis and acute kidney injury (AKI)[1][2].

In Vitro

Lacidipine (0.01-100 μM; 24 h) inhibits HKCs proliferation in vitro in a concentration-dependent manner[1].

Lacidipine (0.01-100 μM; 24 h) protects HKCs against apoptosis induced by ATP depletion and recovery by regulating the caspase-3 pathway[1].

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

Cell Proliferation Assay[1]

Cell Line:	HKC cells
Concentration:	0.01-100 μM
Incubation Time:	24 h
Result:	Exhibited anti-proliferative activity in a concentration-dependent manner.

Apoptosis Analysis[1]

Cell Line:	HKC cells (renal ischemia reperfusion (I/R) model)
Concentration:	1, 10 μM
Incubation Time:	24 h
Result:	AA-induced HKC cells apoptosis, with proportion of early apoptotic cells of 1.47% and 0.30% for 1 and 10 μM dosage, respectively.

Western Blot Analysis[1]

Cell Line:	HKC cells (renal ischemia reperfusion (I/R) model)
Concentration:	1, 10 μM
Incubation Time:	24 h (pretreat)
Result:	Decreased the expression of cyt c of injured cells following ATP depletion and recovery. Significantly increased the expression of the Bcl-2 protein, but decreased the Bax protein.

In Vivo

Lacidipine (0.3, 1.0, 3.0 mg/kg; p.o.; single daily for 10 weeks) shows anti-atherogenic effects in the apoE-deficient mouse, and reduces plasma endothelin concentrations[2].

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

Animal Model:	Female C57BL/6 mice (Homozygous; apoE-deficient; atherosclerosis model)[2].
Dosage:	0.3, 1.0, 3.0 mg/kg
Administration:	Oral gavage; single daily for 10 weeks.
Result:	Induced a significant dose-dependent decrease in plasma endothelin levels. Significantly reduced the mean lesion area in a dose-related manner by 10, 17 and 53% for 0.3, 1.0, 3.0 mg/kg, respectively.

CUSTOMER VALIDATION

- Sci Rep. 2022 Nov 5;12(1):18811.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Zhang A, et al. Lacidipine attenuates apoptosis via a caspase-3 dependent pathway in human kidney cells. Cell Physiol Biochem. 2013;32(4):1040-9.

[2]. Cristofori P, et al. The calcium-channel blocker lacidipine reduces the development of atherosclerotic lesions in the apoE-deficient mouse. J Hypertens. 2000 Oct;18(10):1429-36.

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