Pitavastatin Calcium

Cat. No.: HY-B0144

CAS No.: 147526-32-7

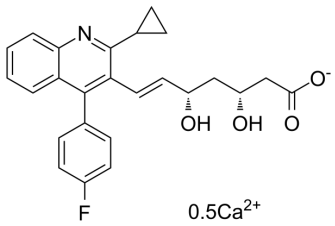
Molecular Formula: $C_{25}H_{23}Ca_{0.5}FNO_4$

Molecular Weight: 440.49

Target: HMG-CoA Reductase (HMGCR); Autophagy; Mitophagy; Apoptosis

Pathway: Metabolic Enzyme/Protease; Autophagy; Apoptosis

Storage: 4°C, sealed storage, away from moisture and light
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (113.51 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2702 mL	11.3510 mL	22.7020 mL
5 mM	0.4540 mL	2.2702 mL	4.5404 mL
10 mM	0.2270 mL	1.1351 mL	2.2702 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 (5.68 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.68 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.68 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pitavastatin Calcium (NK-104 hemicalcium) is a potent hydroxymethylglutaryl-CoA (HMG-CoA) reductase inhibitor. Pitavastatin Calcium (NK-104 hemicalcium) inhibits cholesterol synthesis from acetic acid with an IC_{50} of 5.8 nM in HepG2 cells. Pitavastatin Calcium is an efficient hepatocyte low-density lipoprotein-cholesterol (LDL-C) receptor inducer. Pitavastatin Calcium also possesses anti-atherosclerotic, anti-asthmatic, anti-osteoarthritis, antineoplastic, neuroprotective, hepatoprotective and reno-protective effects[1][2][3][8].

IC_{50} & Target

HMG-CoA Reductase[1]

In Vitro

Pitavastatin Calcium inhibits the growth of a panel of ovarian cancer cells, including those considered most likely to represent HGSOC, grown as a monolayers (IC_{50} = 0.4-5 μM) or as spheroids (IC_{50}=0.6-4 μM)[3].
Pitavastatin Calcium (1 μM; 48 hours) induces apoptosis, evidenced by the increased activity of executioner caspases-3,7 as well as caspase-8 and caspase-9 in Ovcar-8 cells and Ovcar-3 cells[3].
Pitavastatin (1 μM, 48 hours) causes PARP cleavage in Ovcar-8 cells[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[3]

Cell Line:	Ovcar-8 cells
Concentration:	1 μM
Incubation Time:	48 hours
Result:	Induced PARP cleavage.

In Vivo

Pitavastatin Calcium (59 mg/kg; p.o.; twice daily for 28 days) causes significant tumour regression[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	4 week old female NCR Nu/Nu female mice (bearing Ovcar-4 tumours)[3]
Dosage:	59 mg/kg
Administration:	p.o.; twice daily for 28 days
Result:	Caused significant tumour regression.

CUSTOMER VALIDATION

- J Hepatol. 2021 Aug;75(2):363-376.
- Acta Pharm Sin B. 2020 May;10(5):850-860.
- Biochem Pharmacol. 2019 Nov;169:113612.

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REFERENCES

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[2]. Hamano T, et al. Pitavastatin decreases tau levels via the inactivation of Rho/ROCK. Neurobiol Aging. 2012 Oct;33(10):2306-20.

[3]. de Wolf E, et al.Dietary geranylgeraniol can limit the activity of pitavastatin as a potential treatment for drug-resistant ovarian cancer.Sci Rep. 2017 Jul 14;7(1):5410.

[4]. Demir B, et al. The Effects of Pitavastatin on Nuclear Factor-Kappa B and ICAM-1 in Human Saphenous Vein Graft Endothelial Culture. Cardiovasc Ther. 2019 May 2;2019:2549432.

[5]. Hayashi T, et al. A new HMG-CoA reductase inhibitor, pitavastatin remarkably retards the progression of high cholesterol induced atherosclerosis in rabbits. Atherosclerosis. 2004 Oct;176(2):255-63.

[6]. Sahebkar A, et al. A comprehensive review on the lipid and pleiotropic effects of pitavastatin. Prog Lipid Res. 2021 Nov;84:101127.

[7]. Mukhtar RY, et al. Pitavastatin. Int J Clin Pract. 2005 Feb;59(2):239-52.

[8]. Kajinami K, et al. Pitavastatin: efficacy and safety profiles of a novel synthetic HMG-CoA reductase inhibitor. Cardiovasc Drug Rev. 2003 Fall;21(3):199-215.

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