Pitavastatin

Cat. No.: HY-B0144A

CAS No.: 147511-69-1

Molecular Formula: $C_{25}H_{24}FNO_4$

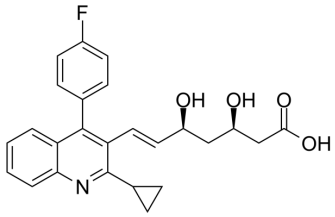
Molecular Weight: 421.46

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

Pathway: Metabolic Enzyme/Protease; Autophagy; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (237.27 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3727 mL	11.8635 mL	23.7270 mL
	5 mM	0.4745 mL	2.3727 mL	4.7454 mL
	10 mM	0.2373 mL	1.1864 mL	2.3727 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 (5.93 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.93 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pitavastatin (NK-104) is a potent hydroxymethylglutaryl-CoA (HMG-CoA) reductase inhibitor. Pitavastatin inhibits cholesterol synthesis from acetic acid with an IC_{50} of 5.8 nM in HepG2 cells. Pitavastatin is an efficient hepatocyte low-density lipoprotein-cholesterol (LDL-C) receptor inducer. Pitavastatin 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 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)[4].
Pitavastatin (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[4].
Pitavastatin (1 μM, 48 hours) causes PARP cleavage in Ovcar-8 cells[4].
Pitavastatin (0.1 and 1 μM; 1 h, then cells incubate with TNF-α for 6 h) increases the expression of ICAM-1 mRNA through suppressing NF-κB pathway in TNF-α-stimulated human saphenous vein endothelial cells[6].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[4]

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

In Vivo

Pitavastatin (59 mg/kg; p.o.; twice daily for 28 days) causes significant tumour regression[4].
Pitavastatin (0.1 mg/kg; p.o; daily for 12 weeks) retards the progression of atherosclerosis formation and improves NO bioavailability by eNOS up-regulation and decrease of O^{2-} in diet induced severe hyperlipidemia rabbit model[7].
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)[4]
Dosage:	59 mg/kg
Administration:	p.o.; twice daily for 28 days
Result:	Caused significant tumour regression.

Animal Model:	Female New Zealand white rabbits (diet induced severe hyperlipidemia)[7]
Dosage:	0.1 mg/kg
Administration:	p.o; daily for 12 weeks
Result:	Retarded the progression of atherosclerosis formation and improved NO bioavailability by eNOS up-regulation and decrease of O^{2-}.

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.
- Proteomics. 2023 May 4;e2300041.

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REFERENCES

[1]. 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.

[2]. 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.

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

[4]. Morikawa S, et al. Relative induction of mRNA for HMG CoA reductase and LDL receptor by five different HMG-CoA reductase inhibitors in cultured human cells. J Atheroscler Thromb. 2000;7(3):138-44.

[5]. Katsuki S, et al. Nanoparticle-mediated delivery of pitavastatin inhibits atherosclerotic plaque destabilization/rupture in mice by regulating the recruitment of inflammatory monocytes. Circulation. 2014 Feb 25;129(8):896-906.

[6]. Tajiri K, et al. Pitavastatin regulates helper T-cell differentiation and ameliorates autoimmune myocarditis in mice. Cardiovasc Drugs Ther. 2013 Oct;27(5):413-24.

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

[8]. 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.

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