Pitavastatin sodium

Cat. No.: HY-B0144B

CAS No.: 574705-92-3

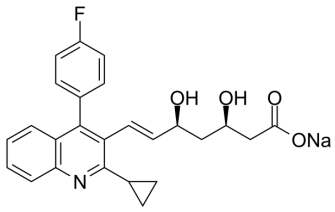
Molecular Formula: $C_{25}H_{23}FNNaO_4$

Molecular Weight: 443.44

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

Pathway: Metabolic Enzyme/Protease; Autophagy; Apoptosis

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Pitavastatin (NK-104) sodium is a potent hydroxymethylglutaryl-CoA (HMG-CoA) reductase inhibitor. Pitavastatin sodium inhibits cholesterol synthesis from acetic acid with an IC_{50} of 5.8 nM in HepG2 cells. Pitavastatin sodium is an efficient hepatocyte low-density lipoprotein-cholesterol (LDL-C) receptor inducer. Pitavastatin sodium 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-}.

REFERENCES

[1]. 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.

[2]. 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.

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[7]. 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.

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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