Product Data Sheet

Product Name: Pitavastatin
Cat. No.: GC11332

Chemical Properties

Cas. No.	147511-69-1		
Chemical Name	(E,3R,5S)-7-[2-cyclopropyl-4-(4-fluorophenyl)quinolin-3-yl]-3,5-dihydroxyhept-6-enoic acid		
SMILES	C1CC1C2=NC3=CC=CC=C3C(=C2C=CC(CC(CC(=O)O)O)O)C4=CC=C(C=C4)F		
Formula	$C_{25}H_{24}FNO_4$	M.Wt	421.46
Solubility	≥ 14.35mg/mL in DMSO	Storage	Store at -20°C,protect from light
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure	Pitavastatin		

Protocol

Cell experiment [1]:

Cell lines	Ovarian cancer Ovcar-8 or Ovcar-3 cells
Preparation Method	Ovarian cancer Ovcar-8 or Ovcar-3 cells were exposed to Pitavastatin (1μM) for 48h.
Reaction Conditions	1μM; 48h

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Applications	Pitavastatin inhibited the growth of cultures of ovarian cancer cells evidenced by the increased activity of executioner caspases-3,7 as well as caspase-8 and caspase-9 in two separate cell lines, and induced PARP cleavage

Animal experiment [2]:

Animal models	BALB/c mice
Preparation Method	The experimental autoimmune myocarditis (EAM) model was established in BALB/c mice by immunization with murine α-myosin heavy chain. Mice were fed Pitavastatin (5mg/kg) or vehicle (control) once daily by gavage feeding for 3 weeks from the beginning of the MyHC-α immunization and lasting to the end of the experiment.
Dosage form	5mg/kg; gavage feeding; daily for 3 weeks.
Applications	Pitavastatin inhibited Th1 and Th17 responses and ameliorated experimental autoimmune myocarditis.

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References:
[1] Wolf E, Abdullah M I, Jones S M, 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.
[2] Tajiri K, Shimojo N, Sakai S, et al. Pitavastatin regulates helper T-cell differentiation and ameliorates autoimmune myocarditis in mice. Cardiovasc Drugs Ther. 2013 Oct;27(5):413-24.

Background

Pitavastatin is a 3-Hydroxymethyl-3-glutaryl-CoA (HMG-CoA) reductase inhibitors. The IC_{50} value of the HMG-CoA reductase activity in the Hep G2 cells for Pitavastatin was 5.8nM[1]. Pitavastatin is clinically employed as an oral statin to lower LDL-cholesterol and prevent cardiovascular events, while also exerting anti-atherosclerotic, anti-asthmatic, anti-osteoarthritis, antineoplastic, neuroprotective, hepatoprotective and reno-protective effects[2][3].

In vitro, M1C transfectant cell line derived from human neuroblastoma BE(2)-M17D cells were exposed to Pitavastatin (0.5, 1, 2, 5, or 10μM) for 0.5, 1, 1.5, 2, or 4 days. Pitavastatin caused a dose-dependent reduction of both total and phosphorylated tau without affecting tau mRNA or cell viability. Maximal clearance was achieved after 36h of 1μM treatment[4]. Human saphenous vein endothelial cells were incubated with 0.1μM or 1μM of Pitavastatin for an hour followed by induction of inflammation by TNF-α. Pitavastatin increased ICAM-1 mRNA expression but did not significantly alter the relative mRNA leve of NF-κB. High-dose Pitavastatin is more cytoprotective since lower LDH levels were obtained in the group of high-dose Pitavastatin compared to low-dose

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Pitavastatin[5]. Ovarian cancer Ovcar-8 or Ovcar-3 cells were exposed to Pitavastatin (1μM) for 48h. Pitavastatin inhibited the growth of cultures of ovarian cancer cells evidenced by the increased activity of executioner caspases-3,7 as well as caspase-8 and caspase-9 in two separate cell lines, and induced PARP cleavage[6].

In vivo, 10 oophorectomized female rabbits were fed with regular diet with or without Pitavastatin (0.1mg/kg per day) for 12 weeks. Pitavastatin retarded the progression of atherosclerosis formation and it improved NO bioavailability by eNOS up-regulation and decrease of O_2^-[7]. Experimental autoimmune myocarditis (EAM) mouse models were fed Pitavastatin (5mg/kg) or vehicle once daily by gavage feeding for 3 weeks. Pitavastatin ameliorated EAM by inhibiting the phosphorylation of signal transducer and activator of transcription STAT3 and STAT4 and suppressing production of Th1 cytokine interferon-γ and Th17 cytokine interleukin-17 from autoreactive $CD4^+$ T cells[8].

References:
[1] Morikawa S, Umetani M, Nakagawa 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] Masana L. Pitavastatin in cardiometabolic disease: therapeutic profile. Cardiovasc Diabetol. 2013 May 30;12(Suppl 1):S2.
[3] Sahebkar A, Kiaie N, Gorabi A M, et al. A comprehensive review on the lipid and pleiotropic effects of Pitavastatin. Prog Lipid Res. 2021 Nov;84:101127.
[4] Hamano T, Yen S H, Gendron T, et al. Pitavastatin decreases tau levels via the inactivation of Rho/ROCK. Neurobiol Aging. 2012 Oct;33(10):2306-20.
[5] Demir B, Onal B, Ozyazgan S, 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.
[6] Wolf E, Abdullah M I, Jones S M, 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.
[7] Hayashi T, Rani J A P, Fukatsu A, 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.
[8] Tajiri K, Shimojo N, Sakai S, et al. Pitavastatin regulates helper T-cell differentiation and ameliorates autoimmune myocarditis in mice. Cardiovasc Drugs Ther. 2013

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