

Product Data Sheet

Product Name: Pitavastatin Calcium

Cat. No.: GC12116

Chemical Properties

Cas. No. 147526-32-7

Chemical Name calcium;(E,3R,5S)-7-[2-cyclopropyl-4-(4-fluorophenyl)quinolin-3-yl]-3,5-dihydroxyhept-6-enoate

SMILES C1CC1C2=NC3=CC=CC=C3C(=C2C=CC(CC(CC(=O)[O-])O)O)C4=CC=C(C=C4)F.C1CC1C2=NC3=CC=CC=C3C(=C2C=CC(CC(CC(=O)[O-])O)O)C4=CC=C(C=C4)F.[Ca+2]

Formula $C_{50}H_{46}CaF_2N_2O_8$

M.Wt 880.98

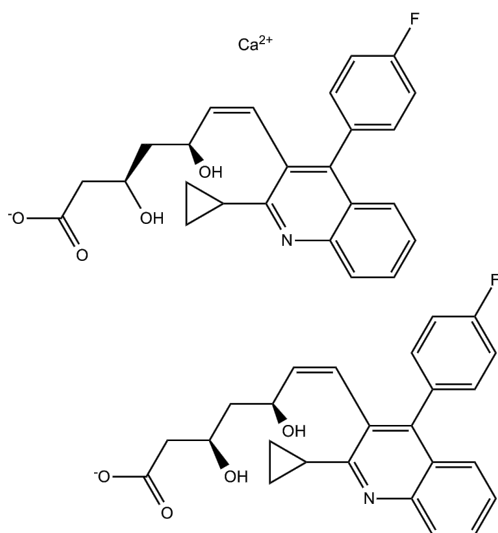
Solubility $\geq 34.85\text{mg/mL}$ in DMSO

Storage 4°C , protect from light

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C 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



Protocol

Cell experiment [1]:

Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

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Cell lines Huh-7 and SMMC7721 liver cancer cell lines

Preparation method The solubility of this compound in DMSO is >34.9mg/mL. General tips for obtaining a higher concentration: Please warm the tube at 37°C for 10 minutes and/or shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Reacting condition 0-20 μ M

Applications In the liver cancer Huh-7 cells and SMMC7721 cells, pitavastatin inhibited cell growth in a dose-dependent way and inhibited colony formation. Pitavastatin significantly arrested the Huh-7 cells at the G1 phase and increased the proportion of sub-G1 phase cells. Pitavastatin induced apoptosis dependent of caspases.

Animal experiment [2]:

Animal models Experimental autoimmune myocarditis (EAM) BALB/c mice

Dosage form 5 mg/kg; 3 weeks from day 0 to day 21; orally

Application In experimental autoimmune myocarditis (EAM) BALB/c mice, pitavastatin reduced the pathophysiological severity of the myocarditis. Pitavastatin inhibited the phosphorylation of STAT3 and STAT4, and suppressed production of Th1 cytokine interferon- γ and Th17 cytokine interleukin-17 from autoreactive CD4⁺T cells in the heart.

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Other notes Please test the solubility of all compounds indoor, and the actual solubility may slightly differ with the theoretical value. This is caused by an experimental system error and it is normal.

References:

[1] You HY1, Zhang WJ1, Xie XM1, et al. Pitavastatin suppressed liver cancer cells in vitro and in vivo. *Onco Targets Ther.* 2016 Aug 29;9:5383-8.

[2]. Tajiri K1, 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 Calcium is a competitive inhibitor of the enzyme HMGCR (HMG-CoA reductase) results in a reduction in LDL cholesterol synthesis. Alternate studies show that pitavastatin can suppress oxygen production in endothelial cells by inhibiting NADPH oxidase. In addition, pitavastatin reduces the expression of eNOS mRNA while increasing the NO dependent response stimulated by acetylcholine and the calcium ionophore, A23187. Furthermore, pitavastatin inhibits the up-regulation of conductance calcium-activated potassium channels by lowering cholesterol levels in cells.

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