

Product Data Sheet

Product Name: Pravastatin sodium

Cat. No.: GC14538

Chemical Properties

Cas. No. 81131-70-6

Chemical Name sodium;(3R,5R)-3,5-dihydroxy-7-[(1S,2S,6S,8S)-6-hydroxy-2-methyl-8-[(2S)-2-methylbutanoyl]oxy-1,2,6,7,8,8a-hexahydronaphthalen-1-yl]heptanoate

SMILES CCC(C)C(=O)OC1CC(C=C2C1C(C(C=C2)C)CCC(CC(CC(=O)[O-])O)O)O.[Na+]

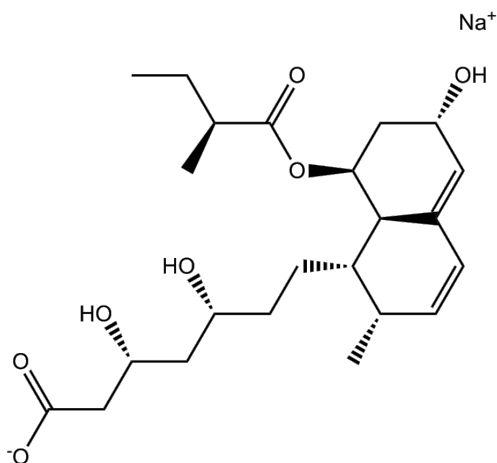
Formula $C_{23}H_{35}O_7.Na$ M.Wt 446.51

Solubility ≥ 13.15 mg/mL in DMSO, ≥ 100.4 mg/mL in EtOH with ultrasonic, ≥ 98.8 mg/mL in Water
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]:

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Cell lines ECFC cells

Preparation Method ECFC cells were seeded into 6-well plates with 5×10^4 cells per well and cultured for 24h. The cell monolayers were scratched with a sterile P1000 pipette tip to create a wound. They were treated with different concentrations (0, 2, 20, 200, and 2000 μ M) of Pravastatin sodium. Phase contrast microscopic images were immediately obtained after scratching and then again after 18h.

Reaction
Conditions 0, 2, 20, 200, and 2000 μ M; 18h

Applications Pravastatin sodium significantly improve the migration of ECFC cells.

Animal experiment [2]:

Animal models Wistar albino rats

Preparation Method Female Wistar albino rats were used and diabetes was induced by intraperitoneal injection of streptozotocin. Thereafter, 10 and 20mg/kg/day doses of Pravastatin sodium were administered by oral gavage to the diabetic rats for 8 weeks. At the end of the experiment, the levels of fasting blood glucose, liver glycogen, and liver enzymes related to carbohydrate metabolism were measured.

Dosage form 10 and 20mg/kg/day for 8 weeks; p.o.

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Applications Pravastatin sodium significantly reduced the liver glycogen synthase and pyruvate kinase levels, but increased the glycogen phosphorylase level in diabetic rats.

References:

- [1] Meyer N, Brodowski L, Richter K, et al. Pravastatin Promotes Endothelial Colony-Forming Cell Function, Angiogenic Signaling and Protein Expression In Vitro. J Clin Med. 2021 Jan 6;10(2):183.
[2] Kaya HK, Demirtas B, Yokus B, et al. Comparative effects of pravastatin and rosuvastatin on carbohydrate metabolism in an experimental diabetic rat model. Acta Pharm. 2024 Mar 30;74(1):117-130.

Background

Pravastatin sodium is a reversible competitive inhibitor of 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase, with an IC_{50} of 5.6 μ M. Pravastatin sodium is commonly used in the research of hypercholesterolemia and various cardiovascular diseases^[1]. HMG-CoAR is located in the cytoplasm of eukaryotic cells and some prokaryotes. The enzyme utilizes NADPH to reduce HMG-CoA to mevalonate, serving as the rate-limiting enzyme in the first stage of cholesterol biosynthesis. Critical catalytic residues of this enzyme include Lys691, Glu559, Asp767, and His866^[2]. Pravastatin sodium binds to the pocket of HMG-CoAR where HMG-CoA would bind, through van der Waals forces, thereby reducing the synthesis of cholesterol^[3].

In vitro, treatment with Pravastatin sodium (1 μ g/mL) alleviates high glucose-induced migration dysfunction in cytotrophoblast (CTB) cells incubated at varying glucose concentrations^[4]. Treatment of endothelial colony-forming cells (ECFCs) with Pravastatin sodium (0-2000 μ M) significantly enhances cell proliferation, migration, and tube formation capabilities. Pravastatin sodium increases the phosphorylation of protein kinase B (AKT) and endothelial nitric oxide synthase (eNOS), as well as the expression levels of heme oxygenase-1 (HO-1), vascular endothelial growth factor A (VEGF-A), and placental growth factor (PlGF). Pravastatin sodium decreases the expression levels of soluble fms-like tyrosine kinase-1 (sFlt-1) and endothelial protein C receptor (Eng)^[5].

In vivo, oral administration of Pravastatin sodium (20mg/kg/day) to diabetic rats for 8 weeks resulted

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in an increase in body weight. Pravastatin sodium significantly reduced the levels of glycogen phosphorylase, lactate dehydrogenase, and glucose-6-phosphatase, thereby improving liver enzyme abnormalities caused by diabetes. Pravastatin sodium also decreased fasting blood glucose levels and increased hepatic glycogen content [6]. Oral treatment with Pravastatin sodium (20mg/kg/day) for 14 days in mice with cardiac fibrosis induced by isoproterenol led to a reduction in CK-MB, serum triglycerides, collagen I and III levels in heart tissue, cardiac inflammatory cells, and cardiac fibrosis levels [7].

References:

- [1] Raasch RH. Pravastatin sodium, a new HMG-CoA reductase inhibitor. DICP. 1991 Apr;25(4):388-94.
- [2] Miziorko HM. Enzymes of the mevalonate pathway of isoprenoid biosynthesis. Arch Biochem Biophys. 2011 Jan 15;505(2):131-43.
- [3] Istvan ES. Structural mechanism for statin inhibition of 3-hydroxy-3-methylglutaryl coenzyme A reductase. Am Heart J. 2002 Dec;144(6 Suppl):S27-32.
- [4] Pantho AF, Mohamed S, Govande JV, et al. Pravastatin Protects Cytotrophoblasts from Hyperglycemia-Induced Preeclampsia Phenotype. Cells. 2024 Sep 13;13(18):1534.
- [5] Meyer N, Brodowski L, Richter K, et al. Pravastatin Promotes Endothelial Colony-Forming Cell Function, Angiogenic Signaling and Protein Expression In Vitro. J Clin Med. 2021 Jan 6;10(2):183.
- [6] Kaya HK, Demirtas B, Yokus B, et al. Comparative effects of pravastatin and rosuvastatin on carbohydrate metabolism in an experimental diabetic rat model. Acta Pharm. 2024 Mar 30;74(1):117-130.
- [7] Rana A, Singh TU, Sharma M, et al. Pravastatin attenuates isoprenaline induced cardiac fibrosis in a mouse model. Biotech Histochem. 2023 Nov;98(8):567-577.

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