Mevastatin

Cat. No.: HY-17408

CAS No.: 73573-88-3

Molecular Formula: $C_{23}H_{34}O_5$

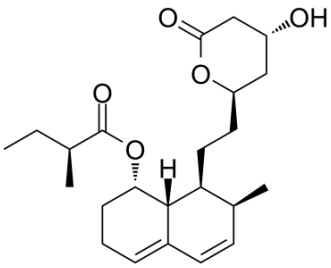
Molecular Weight: 390.51

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

Pathway: Metabolic Enzyme/Protease; Anti-infection; Autophagy; Apoptosis

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (64.02 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5608 mL	12.8038 mL	25.6075 mL
5 mM	0.5122 mL	2.5608 mL	5.1215 mL
10 mM	0.2561 mL	1.2804 mL	2.5608 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 (6.40 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.40 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.40 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Mevastatin (Compactin) is a first HMG-CoA reductase inhibitor that belongs to the statins class. Mevastatin is a lipid-lowering agent, and induces apoptosis, arrests cancer cells in G_0/G_1 phase. Mevastatin also increases endothelial nitric oxide synthase (eNOS) mRNA and protein levels. Mevastatin has antitumor activity and has the potential for cardiovascular diseases treatment[1][2][3].

IC_{50} & Target

HMG-CoA reductase[1][2]
Apoptosis[1]

In Vitro

Mevastatin (0-128 μM; 5 days; Caco-2 cells) treatment causes a dose-dependent decrease in cell number[1].

Mevastatin (32-128 μM; 24-72 hours; Caco-2 cells) treatment causes an early G0/G1 phase and a late G2/M phase cell cyclr arrest[1].
Mevastatin (32-128 μM; 72 hours; Caco-2 cells) treatment causes a down-regulation of cyclin-dependent kinases (cdk) 4 and cdk 6 as well as cyclin D1, while cdk 2 and cyclin E protein levels remained unchanged. Cell cycle inhibitors p21 and p27 are significantly upregulated by Mevastatin[1].
Mevastatin (16-256 μM; Caco-2 cells) treatment induces apoptosis in a dose-dependent manner[1].
Treatment of Neuro2a cells with mevastatin for 24 hours induced neurite outgrowth associated with up-regulation of the neuronal marker protein NeuN. Mevastatin triggers phosphorylation of the key kinases epidermal growth factor receptor (EGFR), ERK1/2, and Akt/protein kinase B. Inhibition of EGFR, PI3K, and the mitogen-activated protein kinase cascade blocks Mevastatin-induced neurite outgrowth[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	Caco-2 cells
Concentration:	0 μM, 8 μM, 16 μM, 32 μM, 64 μM, 128 μM
Incubation Time:	5 days
Result:	Caused a dose-dependent decrease in cell number.

Cell Cycle Analysis[2]

Cell Line:	Caco-2 cells
Concentration:	32 μM, 64 μM, 128 μM
Incubation Time:	24 hours, 48 hours, 72 hours
Result:	Caused a dose-dependent increase of cells in G0/G1 and G2/M phases of the cell cycle.

Western Blot Analysis[2]

Cell Line:	Caco-2 cells
Concentration:	32 μM, 64 μM, 128 μM
Incubation Time:	72 hours
Result:	Resulted in a down-regulation of cyclin-dependent kinases (cdk) 4 and cdk 6 as well as cyclin D1.

In Vivo

Mevastatin (2-20 mg/kg; delivered via ALZET miniosmotic pumps; daily; for 7, 14, or 28 days; wild-type 129-SV/eVTAcBr male mice and eNOS-deficient male mice) treatment increases levels of endothelial nitric oxide synthase (eNOS) mRNA and protein, reduces infarct size, and improves neurological deficits in a dose- and time-dependent manner[2].
The topical infusion of Mevastatin (2.5 pmol/hr) increases bone mass (MRL/MpJ mouse) of isografted bone by increasing bone turnover and, at least in part, by promoting the expression of bone morphogenetic protein-2 (BMP-2) mRNA and receptor activator of NF-κB ligand (RANKL) mRNA[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Wild-type 129-SV/eVTAcBr male mice and eNOS-deficient male mice (18-22 g) with the filament model[2]
Dosage:	2 mg/kg or 20 mg/kg
Administration:	Delivered via 7- or 14-day ALZET miniosmotic pumps implanted subcutaneously; daily; for

	7, 14, or 28 days
Result:	Increased levels of endothelial nitric oxide synthase (eNOS) mRNA and protein, reduced infarct size, and improved neurological deficits in a dose- and time-dependent manner.

CUSTOMER VALIDATION

- Cell Death Dis. 2020 Jan 13;11(1):25.
- Front Cell Dev Biol. 2020 May 28;8:404.
- Sci China Life Sci. 2021 May 27;1-21.
- PLoS Negl Trop Dis. 2019 Aug 20;13(8):e0007681.
- Oncol Lett. 2020 Sep;20(3):2855-2869.

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REFERENCES

[1]. Sugazaki M, Hirotani H, Echigo S, et al. Effects of mevastatin on grafted bone in MRL/MpJ mice. Connect Tissue Res. 2010 Apr;51(2):105-12.

[2]. Evangelopoulos ME, Weis J, Krüttgen A. Mevastatin-induced neurite outgrowth of neuroblastoma cells via activation of EGFR. J Neurosci Res. 2009 Jul;87(9):2138-44.

[3]. Wächtershäuser A, et al. HMG-CoA reductase inhibitor mevastatin enhances the growth inhibitory effect of butyrate in the colorectal carcinoma cell line Caco-2. Carcinogenesis. 2001 Jul;22(7):1061-7.

[4]. Amin-Hanjani S, Stagliano NE, Yamada M, et al. Mevastatin, an HMG-CoA reductase inhibitor, reduces stroke damage and upregulates endothelial nitric oxide synthase in mice. Stroke. 2001 Apr;32(4):980-6.

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