Lovastatin

Cat. No.: HY-N0504

CAS No.: 75330-75-5

Molecular Formula: $C_{24}H_{36}O_5$

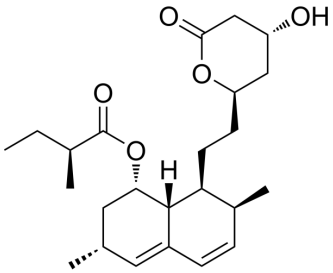
Molecular Weight: 404.54

Target: HMG-CoA Reductase (HMGCR); Autophagy; Ferroptosis

Pathway: Metabolic Enzyme/Protease; Autophagy; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (247.19 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4719 mL	12.3597 mL	24.7194 mL
5 mM	0.4944 mL	2.4719 mL	4.9439 mL
10 mM	0.2472 mL	1.2360 mL	2.4719 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 (6.18 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.18 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.18 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Lovastatin is a cell-permeable HMG-CoA reductase inhibitor used to lower cholesterol.

IC_{50} & Target

HMG-CoA reductase[1]

In Vitro

Lovastatin (10 μM; 72 hours) efficiently reduces viability of HepG2 cells[2].
Lovastatin (10 μM; 48 hours) induces apoptosis in HepG2 cells[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	HepG2 cells
Concentration:	10 μM
Incubation Time:	72 hours
Result:	Efficiently reduced viability of HepG2 cells.

In Vivo

Lovastatin is an inactive lactone that is hydrolyzed in the liver to an active f3-hydroxyacid form. This principal metabolite is the inhibitor of the enzyme HMG-CoA reductase. The K_i is 1 nM. Lovastatin and its β-hydroxyacid metabolite are highly bound to human plasma proteins. Lovastatin crosses the blood-brain and placental barriers[3]. Lovastatin produces a profound reduction of apolipoprotein-B-containing lipoproteins, especially LDL cholesterol and, to a lesser extent, plasma triglycerides, and a small increase in HDL cholesterol[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2023 May 10;8(1):183.
- Cell Metab. 2020 Apr 7;31(4):741-754.e5.
- Nat Commun. 2023 May 26;14(1):3050.
- Nucleic Acids Res. 2023 Sep 22;gkad759.
- Redox Biol. 2022 Feb;49:102207.

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REFERENCES

[1]. Alberts AW, et al. Discovery, biochemistry and biology of lovastatin. Am J Cardiol. 1988 Nov 11;62(15):10J-15J.

[2]. Kah J, et al. Selective induction of apoptosis by HMG-CoA reductase inhibitors in hepatoma cells and dependence on p53 expression. Oncol Rep. 2012 Sep;28(3):1077-83.

[3]. Frishman WH, et al. Lovastatin: an HMG-CoA reductase inhibitor for lowering cholesterol. Med Clin North Am. 1989 Mar;73(2):437-48.

[4]. Tobert JA, et al. Lovastatin and beyond: the history of the HMG-CoA reductase inhibitors. Nat Rev Drug Discov. 2003 Jul;2(7):517-26.

[5]. Ifergan I, et al. Statins reduce human blood-brain barrier permeability and restrict leukocyte migration: relevance to multiple sclerosis. Ann Neurol. 2006 Jul;60(1):45-55.

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