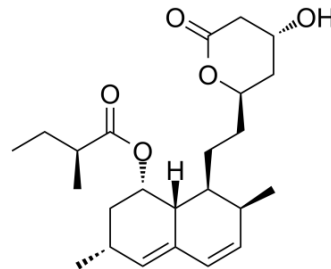


Lovastatin

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	1 mg	5 mg	10 mg
	Concentration			
	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

- 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
- 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
- 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.

IC₅₀ & 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 μ M
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

- Cell Metab. 2020 Apr 7;31(4):741-754.e5.
- Cell Death Dis. 2019 Apr 11;10(4):327.
- Front Cell Dev Biol. 2020 May 28;8:404.
- PLoS Negl Trop Dis. 2019 Aug 20;13(8):e0007681.
- Cell Cycle. 2019 Dec;18(23):3337-3350.

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- [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.

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

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