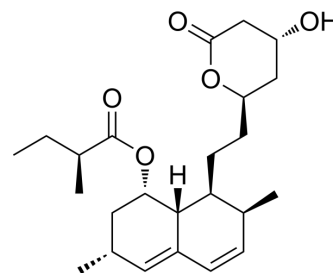


## Lovastatin

Cat. No.:	HY-N0504
CAS No.:	75330-75-5
Molecular Formula:	C <sub>24</sub> H <sub>36</sub> O <sub>5</sub>
Molecular Weight:	404.54
Target:	HMG-CoA Reductase (HMGCR); Autophagy; Ferroptosis
Pathway:	Metabolic Enzyme/Protease; Autophagy; Apoptosis
Storage:	<div> <div>Powder</div> <div>-20°C    3 years</div> <div>4°C    2 years</div> </div> <div> <div>In solvent</div> <div>-80°C    6 months</div> <div>-20°C    1 month</div> </div>



### SOLVENT & SOLUBILITY

#### In Vitro

DMSO : ≥ 100 mg/mL (247.19 mM)  
 \* "≥" means soluble, but saturation unknown.

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	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.
IC <sub>50</sub> & Target	HMG-CoA reductase <sup>[1]</sup>
In Vitro	Lovastatin (10 μM; 72 hours) efficiently reduces viability of HepG2 cells <sup>[2]</sup> . ?Lovastatin (10 μM; 48 hours) induces apoptosis in HepG2 cells <sup>[2]</sup> .

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

Cell Viability Assay<sup>[2]</sup>

Cell Line:	HepG2 cells
Concentration:	10 $\mu$ 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  $\beta$ -hydroxyacid metabolite are highly bound to human plasma proteins. Lovastatin crosses the blood-brain and placental barriers<sup>[3]</sup>. 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<sup>[4]</sup>.

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.

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

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: [tech@MedChemExpress.com](mailto:tech@MedChemExpress.com)

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA