Atorvastatin

Cat. No.: HY-B0589

CAS No.: 134523-00-5

Molecular Formula: $C_{33}H_{35}FN_2O_5$

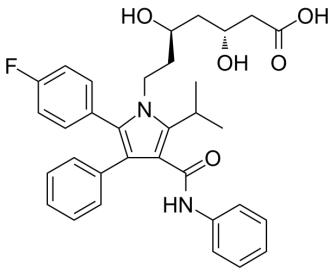
Molecular Weight: 558.64

Target: HMG-CoA Reductase (HMGCR); Autophagy

Pathway: Metabolic Enzyme/Protease; Autophagy

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 : 50 mg/mL (89.50 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.7901 mL	8.9503 mL	17.9006 mL
5 mM	0.3580 mL	1.7901 mL	3.5801 mL
10 mM	0.1790 mL	0.8950 mL	1.7901 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 (4.48 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.48 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Atorvastatin is an orally active HMG-CoA reductase inhibitor, has the ability to effectively decrease blood lipids. Atorvastatin inhibits human SV-SMC proliferation and invasion with IC_{50}s of 0.39 μM and 2.39 μM, respectively[1][2][3].

In Vitro

Atorvastatin treatment decreases apoptosis of myocardial cells by down-regulating GRP78, caspase-12 and CHOP expression in myocardial cells after myocardial infarction, and the endoplasmic reticulum (ER) stress is activated in response to heart failure and angiotensin II (Ang II) stimulation[4].

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

In Vivo

Atorvastatin (20-30 mg/kg; oral gavage; once a day; for 28 days; $ApoE^{-/-}$ mice) treatment significantly reduces endoplasmic reticulum (ER) stress signaling proteins, the number of apoptotic cells, and the activation of Caspase12 and Bax in the Ang II-

induced ApoE-/- mice. Proinflammatory cytokines such as IL-6, IL-8, IL-1β are all remarkably inhibited after Atorvastatin treatment[5].

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

Animal Model:	Forty 8-week-old ApoE$^{-/-}$ mice induced with angiotensin II (Ang II)[5]
Dosage:	20 mg/kg, 30 mg/kg
Administration:	Oral gavage; once a day; for 28 days
Result:	Significantly reduced ER stress signaling proteins, the number of apoptotic cells, and the activation of Caspase12 and Bax in the Ang II-induced ApoE$^{-/-}$ mice. Proinflammatory cytokines such as IL-6, IL-8, IL-1β were all remarkably inhibited.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2023 Dec 25;8(1):457.
- Mol Cell. 2021 Jul 1;81(13):2736-2751.e8.
- Cancer Lett. 2022 Oct 19;215976.
- Cell Death Dis. 2021 May 13;12(5):482.
- Arterioscler Thromb Vasc Biol. 2022 May;42(5):644-658.

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REFERENCES

[1]. Nawrocki, J.W., et al., Reduction of LDL cholesterol by 25% to 60% in patients with primary hypercholesterolemia by atorvastatin, a new HMG-CoA reductase inhibitor. Arterioscler Thromb Vasc Biol, 1995. 15(5): p. 678-82.

[2]. Santodomingo-Garzón T, et al. Atorvastatin inhibits inflammatory hypernociception. Br J Pharmacol. 2006 Sep;149(1):14-22.

[3]. Turner NA, et al. Comparison of the efficacies of five different statins on inhibition of human saphenous vein smooth muscle cell proliferation and invasion. J Cardiovasc Pharmacol. 2007 Oct;50(4):458-61.

[4]. Song XJ, et al. Atorvastatin inhibits myocardial cell apoptosis in a rat model with post-myocardial infarction heart failure by downregulating ER stress response. Int J Med Sci. 2011;8(7):564-72.

[5]. Li Y, et al. Inhibition of endoplasmic reticulum stress signaling pathway: A new mechanism of statins to suppress the development of abdominal aortic aneurysm. PLoS One. 2017 Apr 3;12(4):e0174821.

[6]. Ming-Bai Hu, et al. Atorvastatin induces autophagy in MDA-MB-231 breast cancer cells. Ultrastruct Pathol. Sep-Oct 2018;42(5):409-415.

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