Atorvastatin hemicalcium salt

Cat. No.: HY-17379

CAS No.: 134523-03-8

Molecular Formula: $C_{33}H_{34}Ca_{0.5}FN_2O_5$

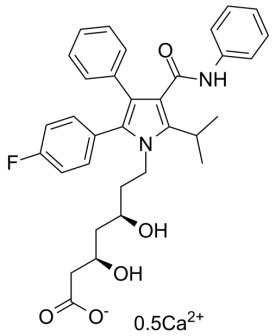
Molecular Weight: 577.67

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

Pathway: Metabolic Enzyme/Protease; Autophagy; Apoptosis

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (86.55 mM)

H_2O : < 0.1 mg/mL (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.7311 mL	8.6555 mL	17.3109 mL
5 mM	0.3462 mL	1.7311 mL	3.4622 mL
10 mM	0.1731 mL	0.8655 mL	1.7311 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 (4.33 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.33 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Atorvastatin hemicalcium salt (CI-981) is an orally active 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitor, has the ability to effectively decrease blood lipids. Atorvastatin hemicalcium salt 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].

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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

- Mol Cell. 2021 Jul 1;81(13):2736-2751.e8.
- Oncogene. 2022 Oct 31.
- Cancer Lett. 2022 Oct 19;215976.
- Arterioscler Thromb Vasc Biol. 2022 May;42(5):644-658.
- Cell Death Dis. 2021 May 13;12(5):482.

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REFERENCES

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

[2]. 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.

[3]. 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.

[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.

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