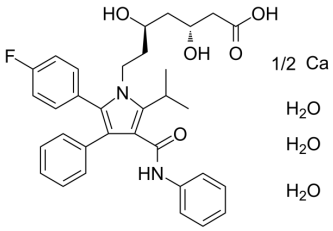
Atorvastatin hemicalcium trihydrate

Cat. No.:	HY-B0589E
CAS No.:	344920-08-7
Molecular Formula:	$C_{33}H_{35}FN_2O_5 \cdot {}_1/_2Ca \cdot {}_3H_2O$
Molecular Weight:	632.73
Target:	HMG-CoA Reductase (HMGCR); Autophagy
Pathway:	Metabolic Enzyme/Protease; Autophagy
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Atorvastatin hemicalcium trihydrate is an orally active HMG-CoA reductase inhibitor, has the ability to effectively decrease blood lipids. Atorvastatin hemicalcium trihydrate 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 hemicalcium trihydrate 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) hemicalcium trihydrate 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.
- Arterioscler Thromb Vasc Biol. 2022 May;42(5):644-658.
- Cell Death Dis. 2021 May 13;12(5):482.

- Front Cell Dev Biol. 2022 Mar 3;10:806081.
- Biotechnol Bioeng. 2021 Sep 3.

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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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