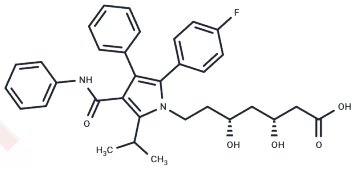
Data Sheet (Cat.No.T20765)

TargetMol

Atorvastatin

Chemical Properties



CAS No. : 134523-00-5

Formula: C33H35FN2O5

Molecular Weight: 558.64

Appearance: no data available

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year

Biological Description

Description	Atorvastatin, an orally active HMG-CoA reductase inhibitor, effectively lowers blood lipids by activating liver cytochrome p450 to accelerate metabolism. Atorvastatin inhibits the proliferation and invasion of human SV-SMC cells with IC50 values of 0.39 μM and 2.39 μM, respectively. [Atorvastatin] combined with clopidogrel may lead to increased thrombotic events in patients.
Targets(IC50)	HMG-CoA Reductase,Autophagy
In vitro	In the atorvastatin group, myocardial cells were lined up more orderly and myocardial fibrosis level was decreased compared to the model group. The expressions of GRP78, caspase-12 and CHOP in myocardial cells were decreased in atorvastatin group. Moreover, in the atorvastatin-treated group the cell apoptosis rate was reduced and the endoplasmic reticulum (ER) stress was activated in response to heart failure and angiotensin II (Ang II) stimulation[1]
In vivo	Higher dose of atorvastatin can effectively suppress the development and progression of AAA induced by Ang II or CaCl2. Mechanistically, the activation of ER stress and inflammatory response were found involved in Ang II-induced AAA formation. The atorvastatin infusion 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, compared with mice treated by Ang II alone. Furthermore, proinflammatory cytokines such as IL-6, IL-8, IL-1β were all remarkably inhibited after atorvastatin treatment. In vitro, the inhibitory effect of simvastatin on the ER stress signal pathway could be observed in both vascular smooth muscle cells and macrophages, and these inhibitory effects of statin were in a dose-dependent manner. In addition, apoptosis was induced with Ang II treatment. The maximal inhibitory effect of simvastatin on apoptosis was observed at 10 μmol/l.

Solubility Information

Solubility	DMSO: 80 mg/mL (143.2 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	1.7901 mL	8.9503 mL	17.9006 mL
5 mM	0.358 mL	1.7901 mL	3.5801 mL
10 mM	0.179 mL	0.895 mL	1.7901 mL
50 mM	0.0358 mL	0.179 mL	0.358 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

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.
Yang D, Fan Y, Xiong M, et al.Loss of renal tubular G9a benefits acute kidney injury by lowering focal lipid accumulation via CES1.EMBO reports.2023: e56128.
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.
Zhang W, Chen L, Liu J, et al.Inhibition of autophagy-related protein 7 enhances anti-tumor immune response and improves efficacy of immune checkpoint blockade in microsatellite instability colorectal cancer.Journal of Experimental & Clinical Cancer Research.2024, 43(1): 1-19.
Rongcai Jiang , Shiguang Zhao, Renzhi Wang ,et al. Safety and Efficacy of Atorvastatin for Chronic Subdural Hematoma in Chinese Patients: A Randomized ClinicalTrial.[J]. Jama Neurology, 2018.
In vivo vulnerabilities to GPX4 and HDAC inhibitors in drug-persistent versus drug-resistant BRAFV600E lung adenocarcinoma