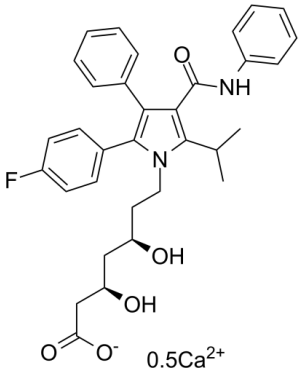
Data Sheet

Product Name:	Atorvastatin (hemicalcium salt)
Cat. No.:	CS-1023
CAS No.:	134523-03-8
Molecular Formula:	C33H34Ca0.5FN2O5
Molecular Weight:	577.67
Target:	Autophagy; Ferroptosis; HMG-CoA Reductase (HMGCR)
Pathway:	Apoptosis; Autophagy; Metabolic Enzyme/Protease
Solubility:	DMSO : ≥ 50 mg/mL (86.55 mM); H2O : < 0.1 mg/mL (insoluble)



BIOLOGICAL ACTIVITY:

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]. **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].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [2]Briefly, SV-SMC from 5 different patients are seeded into 24-well cell culture plates at a density of 1×10^4 cells per well in full growth medium. Cells are incubated overnight and then quiesced in serum free medium for 3 days before transfer to full growth medium (10% FCS) containing 5 different statins (simvastatin, atorvastatin, fluvastatin, lovastatin, and pravastatin)at a range of concentrations. All statins are tested on cells from each individual patient. Medium and drugs are replaced after 2 days, and viable cell numbers are determined in triplicate wells after 4 days using Trypan Blue and a hemocytometer. The increase in cell number is calculated by subtracting the starting cell number (day 0) from the final cell number (day 4). Data are then normalized to control values (no statin) to correct for differences in proliferation rates between cells from different patients. **Animal Administration:** Atorvastatin is formulated in saline.[1]To investigate the effect of atorvastatin on lipopolysaccharide (LPS)-induced inflammatory hypernociception, mice are pretreated orally with either atorvastatin, at doses of 1, 3, 10, 30 and 90 mg/kg or vehicle (PBS) once a day for 3 consecutive days. At 2 h after the last dose of atorvastatin, mice receive an i.pl. injection of LPS (100 ng/paw) or saline (vehicle for LPS). The animals are also treated with atorvastatin (30 mg/kg) for 1 or 2 days before LPS challenge. The hypernociceptive responses are assessed 0.5, 1, 3, 5, 7 and 24 h after LPS or saline i.pl. injections.

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.

CAIndexNames:

1H-Pyrrole-1-heptanoic acid, 2-(4-fluorophenyl)-β,δ-dihydroxy-5-(1-methylethyl)-3-phenyl-4-[(phenylamino)carbonyl]-, calcium salt (2:1), (βR,δR)-

SMILES:

FC1=CC=C(C2=C(C3=CC=CC=C3)C(C(NC4=CC=CC=C4)=O)=C(C(C)C)N2CC[C@@H](O)C[C@@H](O)CC([O-])=O)C=C1.[0.5Ca2+]

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