

Product Data Sheet

Product Name: Atorvastatin

Cat. No.: GC35419

Chemical Properties

Cas. No. 134523-00-5

SMILES O=C(C(C(C1=CC=CC=C1)=C(C2=CC=C(F)C=C2)N3CC[C@@H](O)C[C@@H](O)CC(O)=O)=C3C(C)C)NC4=CC=CC=C4

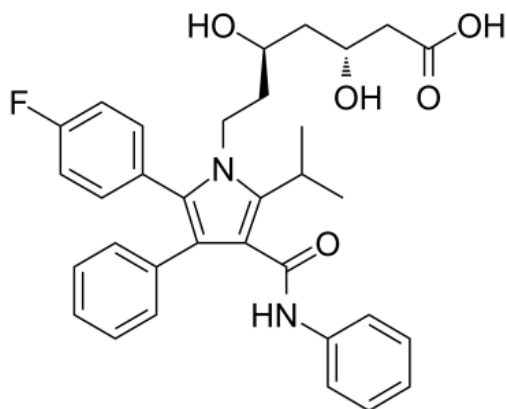
Formula $C_{33}H_{35}FN_2O_5$ M.Wt 558.64

Solubility DMSO: 100 mg/mL (179.01 mM) Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines	Primary rat nucleus pulposus cells
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Preparation Method	Primary rat nucleus pulposus cells were isolated and cultured, and cell viability after exposure to different concentrations was measured using CCK-8. For subsequent experiments, cells were pretreated with Atorvastatin before tumor necrosis factor- α stimulation. Extracellular-matrix metabolism, NLRP3-inflammasome, and NF- κ B-related markers were analyzed using quantitative PCR, western blotting, and immunofluorescence, while autophagic flux was evaluated using mRFP-GFP-LC3 and transmission electron microscopy.
Reaction Conditions	10 and 20 μ M; 1h pretreatment
Applications	Atorvastatin at 20 μ M attenuated tumor necrosis factor- α -induced MMP3, MMP13, ADAMTS4, and Cox-2 expression and restored collagen II and aggrecan expression. NLRP3, cleaved caspase-1, and GSDMD-NT decreased by 52%, 65%, and 48%, respectively, while the LC3-II/LC3-I ratio and autophagic flux increased.
Animal experiment [2]:	
Animal models	Mouse model of lipopolysaccharide-induced depressive-like behavior
Preparation Method	Mice received oral Atorvastatin or vehicle once daily for 7 days and were then given a single intraperitoneal lipopolysaccharide injection. Forced-swim, tail-suspension, and open-field tests were performed 24h later; tumor necrosis factor- α , brain-derived neurotrophic factor, glutathione, and malondialdehyde were then measured in the hippocampus and prefrontal cortex.
Dosage form	1 and 10mg/kg/day; p.o.; 7 days

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Applications	Atorvastatin at 1 and 10mg/kg/day prevented the lipopolysaccharide-induced increase in immobility in the forced-swim and tail-suspension tests without altering open-field locomotion. It also prevented tumor necrosis factor- α elevation, brain-derived neurotrophic factor reduction, enhanced lipid peroxidation, and glutathione loss in the hippocampus and prefrontal cortex.
References: [1] Chen J, Yan J, Li S, Zhu J, Zhou J, Li J, et al. Atorvastatin inhibited TNF-α induced matrix degradation in rat nucleus pulposus cells by suppressing NLRP3 inflammasome activity and inducing autophagy through NF-κB signaling. Cell Cycle. 2021;20(20):2160-2173. doi:10.1080/15384101.2021.1973707. [2] Taniguti EH, Ferreira YS, Stupp IJV, Fraga-Junior EB, Mendonça CB, Rossi FL, et al. Atorvastatin prevents lipopolysaccharide-induced depressive-like behaviour in mice. Brain Res Bull. 2019;146:279-286. doi:10.1016/j.brainresbull.2019.01.018.	

Background

Atorvastatin is a potent synthetic HMG-CoA reductase inhibitor with IC₅₀ values of 7.6nM against the rat liver enzyme and 14.3nM against the human enzyme prepared from HepG2 cells^[1]. Atorvastatin competitively inhibits HMG-CoA reductase, suppresses the mevalonate-cholesterol biosynthetic pathway, and thereby modulates downstream lipid, inflammatory, and cell-survival signaling^{[1][3][4]}. Atorvastatin is used in research on lipid metabolism, inflammatory responses, intervertebral-disc degeneration, and neuroprotection^{[2][3][4][5][6][7]}.

In vitro, HepG2 cells treated with Atorvastatin (0.5 μ M; 24h) showed a 42% reduction in apolipoprotein B secretion, associated with enhanced intracellular apolipoprotein B degradation^[2]. In PMA-differentiated THP-1 cells, Atorvastatin pretreatment (0-100 μ M; 24h), followed by lipopolysaccharide stimulation (0-100 μ g/mL; 24h), reduced interleukin-1 β and tumor necrosis factor- α release and attenuated prostaglandin E₂, reactive oxygen species, and nitric oxide production; at 100 μ M, however, prostaglandin E₂ increased relative to lower-concentration responses^[3]. In primary

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rat nucleus pulposus cells, Atorvastatin (10 and 20 μ M; 1h pretreatment) followed by tumor necrosis factor- α (50ng/mL; 24h) reduced matrix-catabolic and inflammatory proteins, while Atorvastatin (20 μ M; 1h pretreatment) decreased NLRP3, cleaved caspase-1, and GSDMD-NT by 52%, 65%, and 48%, respectively, and increased autophagic flux^[4].

In vivo, rats received Atorvastatin (10mg/kg/day; oral administration once daily for 7 days) before intranasal MPTP exposure; Atorvastatin prevented short-term-memory impairment and depressive-like behavior and reduced motor deficits and substantia-nigra dopaminergic-neuron loss assessed on day21^[5]. In mice, Atorvastatin (0.1mg/kg; single oral administration; behavioral assessment 1h later) produced an antidepressant-like response that was abolished by serotonin depletion or blockade of 5-HT_{1A} or 5-HT_{2A/2C} receptors^[6]. In a lipopolysaccharide-induced depressive-like behavior model, mice received Atorvastatin (1 and 10mg/kg/day; oral administration once daily for 7 days), followed by lipopolysaccharide (0.5mg/kg; single intraperitoneal injection; assessment 24h later). Atorvastatin prevented increased immobility without altering locomotor activity and prevented tumor necrosis factor- α elevation, brain-derived neurotrophic factor reduction, lipid peroxidation, and glutathione depletion in the hippocampus and prefrontal cortex^[7].

References:

- [1] Bergstrom JD, Bostedor RG, Rew DJ, Geissler WM, Wright SD, Chao YS. Hepatic responses to inhibition of 3-hydroxy-3-methylglutaryl-CoA reductase: a comparison of atorvastatin and simvastatin. *Biochim Biophys Acta*. 1998;1389(3):213-221. doi:10.1016/S0005-2760(97)00182-3.
- [2] Wilcox LJ, Barrett PHR, Huff MW. Differential regulation of apolipoprotein B secretion from HepG2 cells by two HMG-CoA reductase inhibitors, atorvastatin and simvastatin. *J Lipid Res*. 1999;40(6):1078-1089. PMID:10357840.
- [3] McFarland AJ, Davey AK, McDermott CM, Grant GD, Lewohl J, Anoopkumar-Dukie S. Statins reduce lipopolysaccharide-induced cytokine and inflammatory mediator release in an in vitro model of microglial-like cells. *Mediators Inflamm*. 2017;2017:2582745. doi:10.1155/2017/2582745.
- [4] Chen J, Yan J, Li S, Zhu J, Zhou J, Li J, et al. Atorvastatin inhibited TNF- α induced matrix degradation in rat nucleus pulposus cells by suppressing NLRP3 inflammasome activity and inducing autophagy through NF- κ B signaling. *Cell Cycle*. 2021;20(20):2160-2173. doi:10.1080/15384101.2021.1973707.

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[5] Castro AA, Wiemes BP, Matheus FC, Lapa FR, Viola GG, Santos AR, et al. Atorvastatin improves cognitive, emotional and motor impairments induced by intranasal 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine administration in rats, an experimental model of Parkinson's disease. Brain Res. 2013;1513:103-116. doi:10.1016/j.brainres.2013.03.029.

[6] Ludka FK, Constantino LC, Dal-Cim T, Binder LB, Zomkowski A, Rodrigues AL, Tasca CI. Atorvastatin evokes a serotonergic system-dependent antidepressant-like effect in mice. Pharmacol Biochem Behav. 2014;122:253-260. doi:10.1016/j.pbb.2014.04.005.

[7] Taniguti EH, Ferreira YS, Stupp IJV, Fraga-Junior EB, Mendonça CB, Rossi FL, et al. Atorvastatin prevents lipopolysaccharide-induced depressive-like behaviour in mice. Brain Res Bull. 2019;146:279-286. doi:10.1016/j.brainresbull.2019.01.018.

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