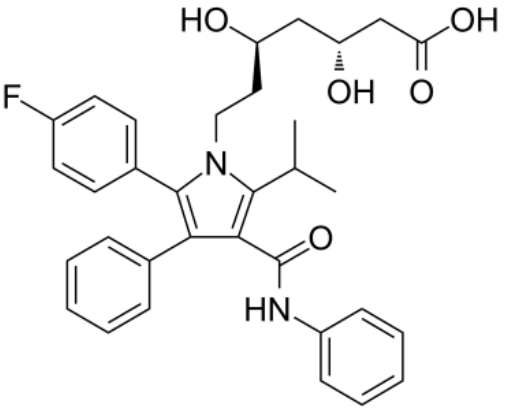
Product Data Sheet

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

Product Data Sheet

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-κB-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 20μM; 1h pretreatment
Applications	Atorvastatin at 20μM 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

Product Data Sheet

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 IC_{50} 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 (0.5μM; 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 (0-100μM; 24h), followed by lipopolysaccharide stimulation (0-100μg/mL; 24h), reduced interleukin-1β and tumor necrosis factor-α release and attenuated prostaglandin E_2, reactive oxygen species, and nitric oxide production; at 100μM, however, prostaglandin E_2 increased relative to lower-concentration responses[3]. In primary

rat nucleus pulposus cells, Atorvastatin (10 and 20μM; 1h pretreatment) followed by tumor necrosis factor-α (50ng/mL; 24h) reduced matrix-catabolic and inflammatory proteins, while Atorvastatin (20μM; 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/$_2$C 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-κB signaling. Cell Cycle. 2021;20(20):2160-2173. doi:10.1080/15384101.2021.1973707.

Product Data Sheet

[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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