Product Data Sheet

Product Name: Alirocumab
Cat. No.: GC19948

Chemical Properties

Cas. No.	1245916-14-6		
Formula		M.Wt	146.24 kDa
Solubility		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	HepG2 cells

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Preparation Method	HepG2 cells were seeded in 6-well plates and cultured as aforementioned. the HepG2 cells were first subjected to Alirocumab (10μg/mL) for 2h, following which LPS was added to a final concentration of 1.0μg/mL.
Reaction Conditions	10μg/mL; 2 hours
Applications	Alirocumab decreases the secretion of LPS-induced pro-inflammatory factors (TNFα, IL-1β, and IL-6) while decreasing LPS-induced activation of the p38-MAPK pathway.

Animal experiment [2]:

Animal models	Sprague Dawley male rats
Preparation Method	Adult Sprague Dawley male rats were used. The rats were fed a 12% alcohol liquid diet or an isocaloric control diet. For the alcohol groups, rats received 5% ethanol during a 5-day acclimation period, followed by 12% ethanol for 6 weeks. Control rats received the same amount of calories from a control liquid diet. Alirocumab (50mg/kg) or vehicle was administered weekly via subcutaneous injection during the entire alcohol exposure period.
Dosage form	50mg/kg weekly for 6 weeks; s.c.
Applications	Alirocumab significantly reduced hepatic triglyceride accumulation and attenuated alcohol-induced hepatic injury markers (ALT and AST). Alirocumab also decreased the expression of pro-inflammatory cytokines (TNF-α, IL-1β, IL-22, IL-33, IL-17α, IL-2, MIP-2, and MCP-1) and neutrophil infiltration.

Caution: Product has not been fully validated for medical applications. For research use only.
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References:
[1] Miao M, Zhang XY, Yu HX, et al. Mechanisms underlying the effects of the conditional knockdown of hepatic PCSK9 in attenuating lipopolysaccharide-induced acute liver inflammation. Int J Biol Macromol. 2025 Feb;291:139066.
[2] Lee JS, Mukhopadhyay P, Matyas C, et al. PCSK9 inhibition as a novel therapeutic target for alcoholic liver disease. Sci Rep. 2019 Nov 20;9(1):17167.

Background

Alirocumab is a human monoclonal antibody that can selectively bind to proprotein convertase subtilisin/kexin type 9 (PCSK9) and inhibit its interaction with the low-density lipoprotein receptor (LDLR)[1]. Alirocumab is used for adjuvant treatment of hyperlipidemia and to reduce the risk of cardiovascular events in specific patients [2]. By blocking PCSK9, Alirocumab can prevent the degradation of LDLR, thereby increasing the number of LDLR on the surface of liver cells and enhancing the clearance of low-density lipoprotein cholesterol (LDL-C) in the blood[3]. These mechanisms make Alirocumab a specific drug for patients who cannot achieve the ideal control level with statins alone or who suffer from genetic hypercholesterolemia (such as familial hypercholesterolemia)[4].

In vitro, after HepG2 cells were treated with Alirocumab (10μg/mL) for 2 hours and then incubated with 1.0μg/mL of LPS for 24 hours, Alirocumab reduced the secretion of pro-inflammatory factors (TNFα, IL-1β and IL-6) induced by LPS and simultaneously decreased the activation of the p38-MAPK pathway induced by LPS[5]. When HepG2 cells were treated with Alirocumab (10μg/mL) for 48 hours, the intracellular cholesterol level was significantly decreased, while the synthesis of bile acids (BAs) was increased. In addition, by inhibiting the activity of PCSK9, Alirocumab reduced the lysosomal degradation of PPARα, thereby increasing the expression of PPARα in the nucleus and activating the transcription of CYP7A1, which promoted the conversion of cholesterol into bile acids[6].

In vivo, Alirocumab (50mg/kg) was subcutaneously injected into Sprague-Dawley rats fed with a 35% ethanol liquid diet, once a week for 6 weeks. Alirocumab significantly reduced the expression of 3-nitrotyrosine (3-NT), an oxidative stress marker induced by ethanol, in the brain, decreased the

recruitment of microglia induced by ethanol, and reduced the expression of pro-inflammatory cytokines and chemokines (TNF, CCL2, CXCL3). Alirocumab also alleviated the oxidative stress and inflammation of cerebrovascular endothelial cells induced by ethanol and decreased the expression of adhesion molecules (ICAM1, VCAM1, eSelectin)[7]. When Alirocumab (50mg/kg) was subcutaneously injected into Sprague-Dawley rats fed with a 12% ethanol liquid diet, Alirocumab significantly reduced the markers of ethanol-induced liver cell injury (ALT and AST), decreased the accumulation of triglycerides in liver cells, and improved the expression of genes related to lipid metabolism (fatty acid synthase FAS, PPARα and CPT1)[8].

References:
[1] Kang C. Alirocumab: Pediatric First Approval. Paediatr Drugs. 2024 Jul;26(4):469-474.
[2] Robinson JG, Farnier M, Krempf M, et al. Efficacy and safety of alirocumab in reducing lipids and cardiovascular events. N Engl J Med. 2015 Apr 16;372(16):1489-99.
[3] Roth EM. Alirocumab for low-density lipoprotein cholesterol lowering. Future Cardiol. 2019 Jan;15(1):17-29.
[4] Tomlinson B, Hu M, Zhang Y, et al. Alirocumab for the treatment of hypercholesterolemia. Expert Opin Biol Ther. 2017 May;17(5):633-643.
[5] Miao M, Zhang XY, Yu HX, et al. Mechanisms underlying the effects of the conditional knockdown of hepatic PCSK9 in attenuating lipopolysaccharide-induced acute liver inflammation. Int J Biol Macromol. 2025 Feb;291:139066.
[6] Chen Z, Shao W, Li Y, et al. Inhibition of PCSK9 prevents and alleviates cholesterol gallstones through PPARα-mediated CYP7A1 activation. Metabolism. 2024 Mar;152:155774.
[7] Wagner J, Park LM, Mukhopadhyay P, Mavromatis LA, Hamandi AM, Rosoff DB, Vendruscolo LF, Koob GF, Pacher P, Lohoff FW. PCSK9 inhibition attenuates alcohol-associated neuronal oxidative stress and cellular injury. Brain Behav Immun. 2024 Jul;119:494-506.
[8] Lee JS, Mukhopadhyay P, Matyas C, et al. PCSK9 inhibition as a novel therapeutic target for alcoholic liver disease. Sci Rep. 2019 Nov 20;9(1):17167.

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