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Alirocumab (anti-PCSK9)

Cat. No.:	HY-P9928A
CAS No.:	1245916-14-6
Target:	PCSK9; NOD-like Receptor (NLR); Keap1-Nrf2; HMG Family; NF-κB; CX3CR1
Pathway:	Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κB; Cell Cycle/DNA Damage
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description

Alirocumab (anti-PCSK9) is a human monoclonal antibody. Alirocumab (anti-PCSK9) inhibits PCSK9. Alirocumab (anti-PCSK9) reduces NLRP3 inflammasome, regulates Nrf2/HO-1, HMGB1/NF-κB and Fractalkine/CX3CR1. Alirocumab (anti-PCSK9) increases the ability of the liver to bind LDL-cholesterol (LDL-C) and reduces levels of LDL-C in blood. Alirocumab (anti-PCSK9) improves atherosclerosis and inflammation[1][2][3][4][5][6][7][8][9][10][11].

IC_{50} & Target

HMGB

In Vitro

Alirocumab (anti-PCSK9) (40 μg/mL, 24 h) alleviates basal PCSK9 overexpression in vascular smooth muscle cells (VSMCs) of obese insulin-resistant Zucker rats (OZR)[3].
Alirocumab (anti-PCSK9) (8 μg/mL, 72 h) attenuates Lp(a) secretion in primary human hepatocytes via inhibition of PCSK9[4].
Alirocumab (anti-PCSK9) (10 μg/mL, 24 h) inhibits lipid-induced inflammation in HepG2 cells[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[5]

Cell Line:	HepG2 incubated with 0.5 mM cis-9-octadecenoic acid and 0.25 mM palmitic acid
Concentration:	10 μg/mL
Incubation Time:	24 h
Result:	Decreased PCSK9 protein levels by 65.3%. Attenuated increased IL-6, IL-1β, and TNFα protein levels. Decreased p65-NF-κB phosphorylation. Reduced the phosphorylation levels of AP-1 by 61.0%. Decreased the phosphorylation levels of PI3K and AKT. Decreased the mTOR protein phosphorylation levels by 46.2%.

In Vivo

Alirocumab (anti-PCSK9) (3-10 mg/kg, s.c., 18 weeks) inhibits atherosclerosis, improves the plaque morphology, and enhances the effects of a statin in APOE*3Leiden.CETP mice[6].
Alirocumab (anti-PCSK9) (16 mg/kg/week, s.c., on day 0, day 7, and day 14) boosts antioxidant status and halts inflammation in rat model of sepsis-induced nephrotoxicity via modulation of Nrf2/HO-1, PCSK9/HMGB1/NF-κB/NLRP3 and Fractalkine/CX3CR1 hubs[7].
Alirocumab (anti-PCSK9) (50 mg/kg, s.c., weekly prior to exposure to the liquid diets) attenuates ethanol-induced neuronal

injury in the brain and oxidative stress in rats s[8].
Alirocumab (anti-PCSK9) (1 mg/kg/week, s.c.) activates brown fat, increases hepatic uptake of cholesterol-rich TRL remnants, thereby lowering non-HDL-C, and increases HDL-C levels and cholesterol efflux capacity of HDL, further improving dyslipidemia in APOE*3-Leiden.CETP mice[9].
Alirocumab (anti-PCSK9) (10 mg/kg, s.c., 2 weeks) reduces lipoprotein(a) levels in nonhuman primates by lowering apolipoprotein(a) production rate[10].
Alirocumab (anti-PCSK9) (3-10 mg/kg, i.p., weekly for 16 weeks) reduces RAS, NLRP3 inflammasome, and cholecystokinin in lung tissue of obese mice[11].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male albino Wistar rats model (LPS-intoxicated)[7]
Dosage:	16 mg/kg/week
Administration:	Subcutaneous injection (s.c.), on day 0, day 7, and day 14
Result:	Mitigated LPS-mediated increments in serum creatinine and cystatin C, together with renal contents of both KIM-1 and NGAL. Restored renal NGAL content to its normal values. Boosted mRNA expression levels of both Nrf2 and HO-1 and renal TAC content (2.5, 2, and 3.2-folds, respectively). Produced pronounced hampering in LPS-mediated elevation in mRNA expression levels of PCSK9 and RAGE, along with renal contents of PCSK9 and HMGB1 by 80.9 %, 49.6 %, 53.1 % and 59.8 %, respectively. Resulted in a marked reduction in the protein expression of TLR4, MYD88, and NLRP3, along with mRNA expression levels of NF-ΚB by 62.9 %, 58.1 %, 50.9 %, respectively. Caused remarkable alleviation in LPS-mediated increment in TNF-α, IL-1β, and caspase-1 by 48.5 %, 68.3 % and 58.5 %, respectively. Produced prominent downregulation in mRNA expression levels of CX3CL1 and CX3CR1 by 88.4 % and 87.5 %, respectively. Exhibited prominent elevation in mRNA expression level of Bcl-2 (1.7-folds), along with a marked reduction in both mRNA expression level of Bax and renal caspase-3 content (by 66.7 % and 58.5 %, respectively) . Regressed glomerular and tubular lesions.

CUSTOMER VALIDATION

- Nat Commun. 2023 Oct 28;14(1):6885.

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