Proteins



Product Data Sheet

PCSK9 Protein, Human (HEK293, Fc)

Cat. No.: HY-P73728

Synonyms: Proprotein convertase subtilisin/kexin type 9; NARC-1; PC9; PCSK9

Species: HEK293 Source:

Accession: Q8NBP7/NP_777596.2 (Q31-Q692)

Gene ID: 255738

Molecular Weight: Approximately 97.4 kDa

PROPERTIES	
Biological Activity	The enzyme activity of this recombinant protein is testing in progress, we cannot offer a guarantee yet.
Appearance	Lyophilized powder
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μ g/mL in ddH ₂ O. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background

PCSK9 protein emerges as a pivotal regulator in the intricate orchestration of plasma cholesterol homeostasis. Demonstrating its influence on low-density lipid receptor family members, including the low-density lipoprotein receptor (LDLR), very low-density lipoprotein receptor (VLDLR), apolipoprotein E receptor (LRP1/APOER), and apolipoprotein receptor 2 (LRP8/APOER2), PCSK9 facilitates their degradation within intracellular acidic compartments. Employing a nonproteolytic mechanism, it enhances the hepatic LDLR degradation through a clathrin LDLRAP1/ARH-mediated pathway, possibly impeding LDLR recycling and directing it toward lysosomal degradation. Moreover, PCSK9 exhibits LDLRindependent inhibition of APOB intracellular degradation via the autophagosome/lysosome pathway and plays a role in the disposal of non-acetylated BACE1 intermediates in the early secretory pathway. Notably, it regulates epithelial Na(+) channel (ENaC)-mediated Na(+) absorption by augmenting ENaC proteasomal degradation, and influences neuronal apoptosis through the modulation of LRP8/APOER2 levels and associated anti-apoptotic signaling pathways.

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Tel: 609-228-6898 Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA

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