

## **Product** Data Sheet

## PCSK9 Protein, Human (Biotinylated, HEK293, D374Y, His-Avi)

Cat. No.: HY-P78824

Synonyms: PCSK9; FH3; HCHOLA3; LDLCQ1; NARC1; PC9

Species: Human HEK293 Source:

Accession: Q8NBP7 (Q31-Q692, D374Y)

Gene ID: 255738 **Molecular Weight:** 16&66 kDa

## **PROPERTIES**

Biological Activity	Measured by its binding ability in a functional ELISA.Immobilized Human PCSK9 at 1 $\mu$ g/mL on streptavidin coated plates can bind Anti-PCSK9 antibody. The EC $_{50}$ is 1.433-2.333 ng/mL.
Appearance	Lyophilized powder
Formulation	Lyophilized a 0.22 μm filtered solution of PBS, 6% Trehalose, 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 $\mu g/mL$ in ddH <sub>2</sub> O.
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.

 $\label{lem:caution:Product} \textbf{Caution: Product has not been fully validated for medical applications. For research use only.}$ 

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