PCSK9 Protein, Mouse (660a.a, HEK293, His)

Cat. No.:	HY-P73339
Synonyms:	Proprotein convertase subtilisin/kexin type 9; NARC-1; PC9; PCSK9
Species:	Mouse
Source:	HEK293
Accession:	Q80W65 (Q35-Q694)
Gene ID:	100102
Molecular Weight:	Approximately 19 & 65 kDa

PROPERTIES

AA Sequence

MGTHCSAWLR
W PLLPLLPPLL LLLLLLCPTG AGAQDEDGDY
EELMLALPSQ EDGLADEAAH VATATFRRCS KEAWRLPGTY
IVVLMEETQR LQIEQTAHRL QTRAARRGYV IKVLHIFYDL
FPGFLVKMSS DLLGLALKLP HVEYIEEDSF VFAQSIPWNL
ERIIPAWHQT EEDRSPDGSS QVEVYLLDTS IQGAHREIEG
RVTITDFNSV PEEDGTRFHR QASKCDSHGT HLAGVVSGRD
AGVAKGTSLH SLRVLNCQGK GTVSGTLIGL EFIRKSQLIQ
PSGPLVVLLP LAGGYSRILN AACRHLARTG VVLVAAAGNF
RDDACLYSPA SAPEVITVGA TNAQDQPVTL GTLGTNFGRC
VDLFAPGKDI IGASSDCSTC FMSQSGTSQA AAHVAGIVAR
MLSREPTLTL AELRQRLIHF STKDVINMAW FPEDQQVLTP
NLVATLPPST HETGGQLLCR TVWSAHSGPT RTATATARCA
PEEELLSCSS FSRSGRRRGD WIEAIGGQQV CKALNAFGGE
GVYAVARCCL VPRANCSIHN TPAARAGLET HVHCHQKDHV
LTGCSFHWEV EDLSVRRQPA LRSRRQPGQC VGHQAASVYA
SCCHAPGLEC KIKEHGISGP SEQVTVACEA GWTLTGCNVL
PGASLTLGAY SVDNLCVARV HDTARADRTS GEATVAAAIC
CRSRPSAKAS WVQ

Biological Activity	Measured by its binding ability in a functional ELISA. Immobilized mouse PCSK9 at 10 μg/mL (100 μL/well) can bind human LDLR and the EC_{50} is 0.12 μg/mL.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of 15 mM Tris, 90 mM NaCl, 50% Glycerol, pH 7.5. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.

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	The PCSK9 protein plays a pivotal role in the intricate regulation of plasma cholesterol homeostasis. It engages with members of the low-density lipid receptor family, including low-density lipoprotein receptor (LDLR), very low-density lipoprotein receptor (VLDLR), apolipoprotein E receptor (LRP1/APOER), and apolipoprotein receptor 2 (LRP8/APOER2), facilitating their degradation within intracellular acidic compartments. Operating through a non-proteolytic mechanism, PCSK9 enhances the degradation of hepatic LDLR via a clathrin LDLRAP1/ARH-mediated pathway and may impede LDLR recycling from endosomes to the cell surface, directing it to lysosomes for degradation. Moreover, PCSK9 induces ubiquitination of LDLR, leading to subsequent degradation, and inhibits the intracellular degradation of APOB through the autophagosome/lysosome pathway, independent of LDLR. Additionally, PCSK9 is implicated in the disposal of non-acetylated intermediates of BACE1 in the early secretory pathway, hinders epithelial Na(+) channel (ENaC)-mediated Na(+) absorption by increasing its proteasomal degradation, and modulates neuronal apoptosis by regulating LRP8/APOER2 levels and related anti-apoptotic signaling pathways.

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