10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.622.0609

PCSK9 Blocking Peptide (C-term)

Synthetic peptide
Catalog # BP20995c

Specification

PCSK9 Blocking Peptide (C-term) - Product Information

Primary Accession Q8NBP7

PCSK9 Blocking Peptide (C-term) - Additional Information

Gene ID 255738

Other Names
Proprotein convertase subtilisin/kexin type 9, 3421-, Neural apoptosis-regulated convertase 1, NARC-1, Proprotein convertase 9, PC9, Subtilisin/kexin-like protease PC9, PCSK9, NARC1

Target/Specificity
The synthetic peptide sequence is selected from aa 654-668 of HUMAN PCSK9

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

PCSK9 Blocking Peptide (C-term) - Protein Information

Name PCSK9

Synonyms NARC1

Function
Crucial player in the regulation of plasma

PCSK9 Blocking Peptide (C-term) - Background

Crucial player in the regulation of plasma cholesterol homeostasis. Binds to low-density lipid receptor family members: low density lipoprotein receptor (LDLR), very low density lipoprotein receptor (VLDLR), apolipoprotein E receptor (LRP1/APOER) and apolipoprotein receptor 2 (LRP8/APOER2), and promotes their degradation in intracellular acidic compartments. Acts via a non-proteolytic mechanism to enhance the degradation of the hepatic LDLR through a clathrin LDLRAP1/ARH-mediated pathway. May prevent the recycling of LDLR from endosomes to the cell surface or direct it to lysosomes for degradation. Can induce ubiquitination of LDLR leading to its subsequent degradation. Inhibits intracellular degradation of APOB via the autophagosome/lysosome pathway in a LDLR-independent manner. Involved in the disposal of non-acetylated intermediates of BACE1 in the early secretory pathway. Inhibits epithelial Na(+) channel (ENaC)-mediated Na(+) absorption by reducing ENaC surface expression primarily by increasing its proteasomal degradation. Regulates neuronal apoptosis via modulation of LRP8/APOER2 levels and related anti-apoptotic signaling pathways.

PCSK9 Blocking Peptide (C-term) - References

Chiang L.W.,et al.Patent number WO0131007, 03-MAY-2001.
Ding K.,et al.PLoS ONE 2:E1098-E1098(2007).
Ota T.,et al.Nat. Genet. 36:40-45(2004).
Gregory S.G.,et al.Nature 441:315-321(2006).
Mural R.J.,et al.Submitted (SEP-2005) to the EMBL/GenBank/DDBJ databases.

cholesterol homeostasis. Binds to low-density lipid receptor family members: low density lipoprotein receptor (LDLR), very low density lipoprotein receptor (VLDLR), apolipoprotein E receptor (LRP1/APOER) and apolipoprotein receptor 2 (LRP8/APOER2), and promotes their degradation in intracellular acidic compartments (PubMed:18039658). Acts via a non- proteolytic mechanism to enhance the degradation of the hepatic LDLR through a clathrin LDLRAP1/ARH-mediated pathway. May prevent the recycling of LDLR from endosomes to the cell surface or direct it to lysosomes for degradation. Can induce ubiquitination of LDLR leading to its subsequent degradation (PubMed:18799458, PubMed:17461796, PubMed:18197702, PubMed:22074827). Inhibits intracellular degradation of APOB via the autophagosome/lysosome pathway in a LDLR-independent manner. Involved in the disposal of non-acetylated intermediates of BACE1 in the early secretory pathway (PubMed:18660751). Inhibits epithelial Na(+) channel (ENaC)-mediated Na(+) absorption by reducing ENaC surface expression primarily by increasing its proteasomal degradation. Regulates neuronal apoptosis via modulation of LRP8/APOER2 levels and related anti-apoptotic signaling pathways.

Cellular Location

Cytoplasm. Secreted. Endosome. Lysosome. Cell surface. Endoplasmic reticulum. Golgi apparatus. Note=Autocatalytic cleavage is required to transport it from the endoplasmic reticulum to the Golgi apparatus and for the secretion of the mature protein Localizes to the endoplasmic reticulum in the absence of LDLR and colocalizes to the cell surface and to the endosomes/lysosomes in the

presence of LDLR. The sorting to the cell surface and endosomes is required in order to fully promote LDLR degradation

Tissue Location
Expressed in neuro-epithelioma, colon carcinoma, hepatic and pancreatic cell lines, and in Schwann cells

PCSK9 Blocking Peptide (C-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides