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PCSK9 Antibody (N-term) Blocking Peptide

Synthetic peptide
Catalog # BP7333a

Specification

PCSK9 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession Q8NBP7

PCSK9 Antibody (N-term) Blocking Peptide - 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 used to generate the antibody AP7333a was selected from the N-term region of human PCSK9. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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 Antibody (N-term) Blocking Peptide - Protein Information

Name PCSK9

Synonyms NARC1

Function
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 (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:<a href="http://www.uniprot.org/citations/18799458"

target="_blank">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 Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

PCSK9 Antibody (N-term) Blocking Peptide - Images

PCSK9 Antibody (N-term) Blocking Peptide - Background

PCSK9 is a proprotein convertase belonging to the proteinase K subfamily of the secretory subtilase family. This protein is synthesized as a soluble zymogen that undergoes autocatalytic intramolecular processing in the endoplasmic reticulum. The protein may function as a proprotein convertase. The protein plays a role in cholesterol homeostasis and may have a role in the differentiation of cortical neurons.

PCSK9 Antibody (N-term) Blocking Peptide - References

Abifadel,M., Rabes,J.P. Hum. Mutat. 30 (7), E682-E691 (2009)McNutt,M.C., Kwon,H.J. J. Biol. Chem. 284 (16), 10561-10570 (2009)Shioji,K., Mannami,T. J. Hum. Genet. 49 (2), 109-114 (2004)Abifadel,M., Varret,M. Nat. Genet. 34 (2), 154-156 (2003)