



Proprotein convertase subtilisin/kexin type 9 (PCSK9), LDLR-binding epitope blocking.
Rabbit Antigen Immunoaffinity Purified Polyclonal
Proprotein convertase PC9, Subtilisin/kexin-like protease PC9, Neural apoptosis-regulated convertase 1

BACKGROUND

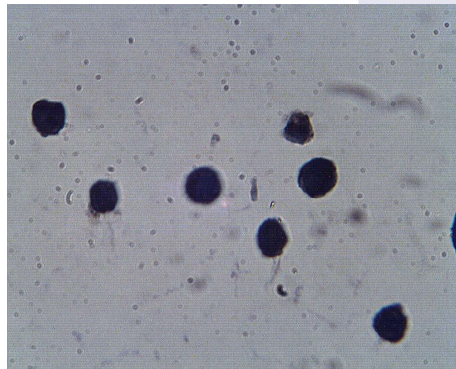
This antibody is made to an epitope that is reported to block the PCSK9-LDLR (low density lipoprotein receptor) interaction. PCSK9 binds to the EGF-A domain of the LDLR and signals LDLR degradation. Reduced LDLR levels result in decreased LDL (low density lipid) metabolism leading to hypercholesterolemia.

Additionally, PCSK9 may be implicated in the differentiation of cortical neurons and may also play a role in cholesterol homeostasis. Defects in PCSK9 gene are the cause of familial hypercholesterolemia 3 (FH3). The protein is thought to play a central role in cholesterol homeostasis

IMMUNOGEN

Synthetic peptide derived from the human PCSK9 protein reported to block PCSK9-LDLR interaction.

Staining of Hep G2 cells with PCSK9 antibody (Cat. No. X2470P) at 2 µg/ml.



ORDERING INFORMATION

CATALOG NUMBER
X2740P

SIZE
100 µg

FORM
Affinity Purified

HOST/CLONE
Rabbit

FORMULATION
Provided as solution in phosphate buffered saline with 0.08% sodium azide

CONCENTRATION
See vial for concentration

ISOTYPE
IgG

APPLICATIONS
Western Blot, Functional epitope reported to block PCSK9-LDLR interaction, ELISA

SPECIES REACTIVITY
Human

ACCESSION NUMBER
Human Q8NBP7

POSITIVE CONTROL/TISSUE EXPRESSION

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

COMMENTS

Antibody can be used for Western blotting (1:400 dilution) and immunocytochemistry (2 µg/ml). Optimal concentration should be evaluated by serial dilutions.

PURIFICATION

Antigen Immunoaffinity Purification

SHIP CONDITIONS

Ship at ambient temperature, freeze upon arrival

STORAGE CUSTOMER

Product should be stored at -20°C. Aliquot to avoid freeze/thaw cycles

STABILITY

Products are stable for one year from purchase when stored properly

REFERENCES

1. Proc Natl Acad Sci U S A. 2003 Feb 4;100(3):928-33. Epub 2003 Jan 27. The secretory proprotein convertase neural apoptosis-regulated convertase 1 (NARC-1): liver regeneration and neuronal differentiation. Seidah NG, et al
2. Nat Genet. 2003 Jun;34(2):154-6. Mutations in PCSK9 cause autosomal dominant hypercholesterolemia. Abifadel et al
3. Hum Mutat. 2005 Nov;26(5):497. Novel mutations of the PCSK9 gene cause variable phenotype of autosomal dominant hypercholesterolemia. Allard D et al
4. Am J Hum Genet. 2006 Mar;78(3):410-22. Epub 2006 Jan 20. A spectrum of PCSK9 alleles contributes to plasma levels of low-density lipoprotein cholesterol. Kotowski IK, et al
5. Clin Genet. 2004 May;65(5):419-22. Mutations in the PCSK9 gene in Norwegian subjects with autosomal dominant hypercholesterolemia. Leren TP.
6. PLoS One. 2007 Oct 31;2(10):e1098. Evidence for positive selection in the C-terminal domain of the cholesterol metabolism gene PCSK9 based on phylogenetic analysis in 14 primate species. Ding K, McDonough SJ, Kullo IJ.
7. J Hum Genet. 2004;49(2):109-14. Epub 2004 Jan 15. Genetic variants in PCSK9 affect the cholesterol level in Japanese. Shioji K, et al
8. Atherosclerosis. 2005 Oct;182(2):331-40. Genetic screening protocol for familial hypercholesterolemia which includes splicing defects gives an improved mutation detection rate. Graham CA et al
9. Cell. 2006 Nov 3;127(3):635-48. Global, in vivo, and site-specific phosphorylation dynamics in signaling networks. Olsen J V et al
10. J Am Coll Cardiol. 2005 May 17;45(10):1611-9. Epub 2005 Apr 21. A common PCSK9 haplotype, encompassing the E670G coding single nucleotide polymorphism, is a novel genetic marker for plasma low-density lipoprotein cholesterol levels and severity of coronary atherosclerosis. Chen SN et al

PRODUCT SPECIFIC REFERENCES