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Produktinformation

- Forschungsprodukte & Biochemikalien
- Zellkultur & Verbrauchsmaterial
- Diagnostik & molekulare Diagnostik
- Laborgeräte & Service

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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PRODUCT INFORMATION

PCSK9 (human, recombinant)

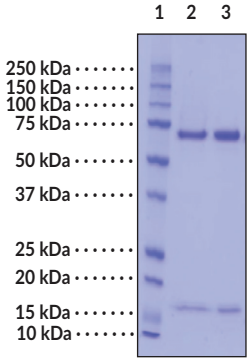
Item No. 20631

Overview and Properties

Synonyms:	NARC-1, Proprotein Convertase Subtilisin Kexin 9
Source:	Active recombinant N-terminal His-tagged enzyme isolated from a HEK293 overexpression system
Amino Acids:	31-692
Uniprot No.:	Q8NBP7
Molecular Weight:	71 kDa (~16 & 63 kDa purified)
Storage:	-20°C (as supplied); avoid freeze/thaw cycles by aliquoting protein
Stability:	≥9 months
Purity:	≥95% estimated by SDS-PAGE
Supplied in:	Lyophilized from PBS, pH 7.4, with 30% sucrose
Protein Concentration:	Recommended reconstitution in serum-free media or sterile buffer to 200 µg/ml
Activity:	***batch specific*** Qualitative biological assay - reduces uptake of LDL-DyLight™ by >30% in Huh7 cells at concentrations above 5 µg/ml

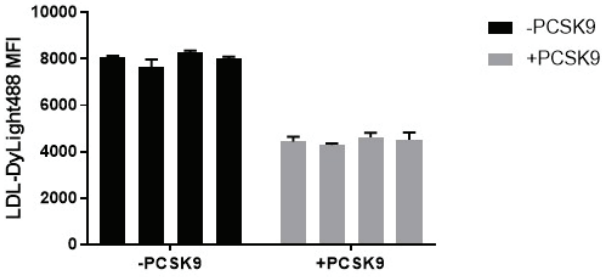
Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Images



Lane 1: MW Markers
Lane 2: PCSK9 (1 µg)
Lane 3: PCSK9 (2 µg)

Representative gel image shown; actual purity may vary between each batch.



PCSK9 inhibits LDL uptake. On four separate plates Huh7 cells were plated and treated with or without 5 µg/ml PCSK9 in media. Sixteen hours later, LDL-DyLight™ 488 was added. After a four-hour incubation the cells were washed, trypsanized, and analyzed by flow cytometry.

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PRODUCT INFORMATION

Description

Proprotein convertase subtilisin kexin 9 (PCSK9) is a member of the subtilisin serine protease family with an important role in lipoprotein metabolism.[1] Mutation in the PCSK9 gene is associated with autosomal dominant hypercholesterolemia, which is characterized by an increase in low density lipoprotein (LDL) cholesterol levels.[2] PCSK9 overexpression in wild-type mice doubles the plasma total cholesterol, possibly through acceleration of the degradation of the LDL receptor.[1,3] PCSK9 mRNA is detected in various tissues such as liver, kidney, lung, spleen, jejunum, ileum, colon, and muscle, with the highest expression in the liver.[4] Human PCSK9 precursor is 692 amino acids in length with an estimated molecular weight of 74 kDa. This proprotein is self-cleaved to form a mature protein at around 63 kDa in the Golgi.[5] Cayman's PCSK9 (human, recombinant) contains the mature protein (~63 kDa) as well as the cleaved portion (~16 kDa).

References

1. Maxwell, K.N., Fisher, E.A., and Breslow, J.L. Overexpression of PCSK9 accelerates the degradation of the LDLR in a post-endoplasmic reticulum compartment. *Proc. Natl. Acad. Sci. USA* **102(6)**, 2069-2074 (2005).
2. Abifadel, M., Varret, M., Rabès, J.-P., *et al.* Mutations in PCSK9 cause autosomal dominant hypercholesterolemia. *Nature Genet.* **34(2)**, 154-156 (2003).
3. Maxwell, K.N. and Breslow, J.L. Adenoviral-mediated expression of Pcsk9 in mice results in a low-density lipoprotein receptor knockout phenotype. *Proc. Natl. Acad. Sci. USA* **101(18)**, 7100-7105 (2004).
4. Seidah, N.G., Benjannet, S., Wickham, L., *et al.* The secretory proprotein convertase neural apoptosis-regulated convertase 1 (NARC-1): Liver regeneration and neuronal differentiation. *Proc. Natl. Acad. Sci. USA* **100(3)**, 928-933 (2003).
5. Maxwell, K.N. and Breslow, J.L. Proprotein convertase subtilisin kexin 9: The third locus implicated in autosomal dominant hypercholesterolemia. *Curr. Opin. Lipidol.* **16**, 167-172 (2005).

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