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Produktinformation

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PRODUCT INFORMATION

HMG-CoA Reductase (human recombinant)

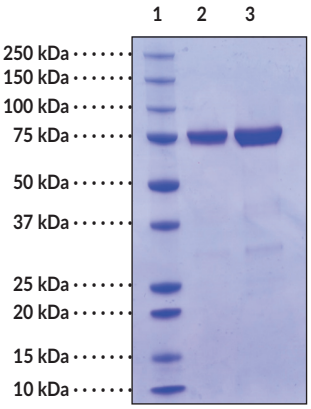
Item No. 14944

Overview

Synonyms:	3-hydroxy-3-methylglutaryl-Coenzyme A Reductase, HMGCR, HMGR
Source:	Recombinant N-terminal GST-tagged protein expressed in *E. coli*
Amino Acids:	426-888 (N-terminal truncation)
Uniprot No.:	P04035
Molecular Weight:	76.5 kDa
Storage:	-80°C (as supplied)
Stability:	≥2 years
Purity:	***batch specific*** (≥85% estimated by SDS-PAGE)
Supplied in:	50 mM Tris, pH 8.0, with 300 mM sodium chloride and 20% glycerol
Activity:	***batch specific***
Specific Activity:	One unit is defined as the amount of enzyme required to convert 1 µmol of NADPH to $NADP^+$ per minute at 37°C in 20 mM sodium phosphate, pH 6.5, 100 mM sodium chloride, and 10 mM DTT, containing 300 µM (R,S)-HMG-CoA and 300 µM NADPH.
Protein Concentration:	***batch specific***

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

Image



Lane 1: MW Markers
Lane 2: HMG-CoA Reductase (2 µg)
Lane 3: HMG-CoA Reductase (4 µg)

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PRODUCT INFORMATION

Description

HMG-CoA reductase (HMGR) is a highly regulated enzyme found on the endoplasmic reticulum (ER) membrane. It is bound to the ER membrane by a multi-pass 339 amino acid N-terminal transmembrane domain while the carboxy terminal catalytic region projects into the cytosol.[1,2] HMGR is controlled by feedback regulation from sterols and non-sterol metabolites derived from mevalonate.[3-4] Binding of cholesterol derived from internalized LDL receptors suppresses HMGR. The enzyme is responsible for catalyzing the rate-limiting step in cholesterol biosynthesis. Mevalonate, which is converted to isopentenyl pyrophosphate, is the building block for cholesterol and non-sterol isoprenoids. The four-electron reduction of HMG-CoA catalyzed by HMGR to form mevalonate is the committed step in the biosynthesis of sterols and isoprenoids.[5] Potent inhibitors of HMGR, collectively called statins, are effective in lowering mortality due to hypercholesterolemia by lowering serum cholesterol levels.[6]

References

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3. Brown, M.S., and Goldstein, J.L. Multivalent feedback regulation of HMG CoA reductase, a control mechanism coordinating isoprenoid synthesis and cell growth. *Journal of Lipid Research* **21(5)**, 505-517 (1980).
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5. Istvan, E.S., and Deisenhofer, J. Structural mechanism for statin inhibition of HMG-CoA reductase. *Science 292(5519)*, 1160-1164 (2001).
6. Brown, W.V. Safety of statins. *Current Opinion in Lipidology* **19(6)**, 558-562 (2008).

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