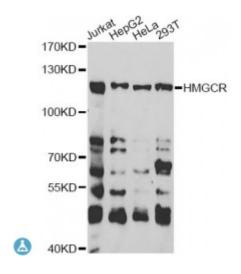


## **Anti-HMGCR Antibody**



**Description** 

HMG-CoA reductase is the rate-limiting enzyme for cholesterol synthesis and is regulated via a negative feedback mechanism mediated by sterols and non-sterol metabolites derived from mevalonate, the product of the reaction catalyzed by reductase. Normally in mammalian cells this enzyme is suppressed by cholesterol derived from the internalization and degradation of low density lipoprotein (LDL) via the LDL receptor. Competitive inhibitors of the reductase induce the expression of LDL receptors in the liver, which in turn increases the catabolism of plasma LDL and lowers the plasma concentration of cholesterol, an important determinant of atherosclerosis. Alternatively spliced transcript variants encoding different isoforms have been found for this gene.

Model STJ115278

**Host** Rabbit

**Reactivity** Human, Rat

**Applications** IF, IHC, WB

Immunogen Recombinant fusion protein containing a sequence corresponding to amino

acids 629-888 of human HMGCR (NP\_000850.1).

**Gene ID** <u>3156</u>

Gene Symbol HMGCR

**Dilution range** WB 1:500 - 1:2000

IHC 1:50 - 1:200 IF 1:50 - 1:200

**Purification** Affinity purification

**Note** For Research Use Only (RUO).

**Protein Name** 3-hydroxy-3-methylglutaryl-coenzyme A reductase HMG-CoA reductase

Molecular Weight 97.476 kDa

**Clonality** Polyclonal

**Conjugation** Unconjugated

**Isotype** IgG

**Formulation** PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Storage Instruction** Store at -20C. Avoid freeze / thaw cycles.

Database Links HGNC:5006OMIM:142910Reactome:R-HSA-191273

Alternative Names 3-hydroxy-3-methylglutaryl-coenzyme A reductase HMG-CoA reductase

**Function** Transmembrane glycoprotein that is the rate-limiting enzyme in cholesterol

biosynthesis as well as in the biosynthesis of nonsterol isoprenoids that are essential for normal cell function including ubiquinone and geranylgeranyl

proteins

Cellular Localization Endoplasmic reticulum membrane

Post-translational N-glycosylated, Deglycosylated by NGLY1 on release from the endoplasmic

**Modifications** reticulum (ER) in a sterol-mediated manner,

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