

**PRODUCT INFORMATION**

<b>Target</b>	LDLR
<b>Synonyms</b>	FH;FHC;FHCL1;LDLCQ2
<b>Description</b>	Recombinant Human LDLR Protein with C-terminal 6×His tag
<b>Delivery</b>	In Stock
<b>Uniprot ID</b>	P01130
<b>Expression Host</b>	HEK293
<b>Tag</b>	C-6×His Tag
<b>Molecular Characterization</b>	LDLR(Ala22-Arg788) 6×His tag
<b>Molecular Weight</b>	The protein has a predicted molecular mass of 85.6 kDa after removal of the signal peptide. The apparent molecular mass of LDLR-His is approximately 100-130 kDa due to glycosylation.
<b>Purity</b>	The purity of the protein is greater than 85% as determined by SDS-PAGE and Coomassie blue staining.
<b>Formulation &amp; Reconstitution</b>	Lyophilized from sterile PBS, pH 7.4. Normally 5% - 8% trehalose is added as protectants before lyophilization. Please see Certificate of Analysis for specific instructions of reconstitution.
<b>Storage &amp; Shipping</b>	Store at -20°C to -80°C for 12 months in lyophilized form. After reconstitution, if not intended for use within a month, aliquot and store at -80°C (Avoid repeated freezing and thawing). Lyophilized proteins are shipped at ambient temperature.
<b>Background</b>	The low density lipoprotein receptor (LDLR) gene family consists of cell surface proteins involved in receptor-mediated endocytosis of specific ligands. Low density lipoprotein (LDL) is normally bound at the cell membrane and taken into the cell ending up in lysosomes where the protein is degraded and the cholesterol is made available for repression of microsomal enzyme 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase, the rate-limiting step in cholesterol synthesis. At the same time, a reciprocal stimulation of cholesterol ester synthesis takes place. Mutations in this gene cause the autosomal dominant disorder, familial hypercholesterolemia. Alternate splicing results in multiple transcript variants.[provided by RefSeq, Sep 2010]
<b>Usage</b>	Research use only



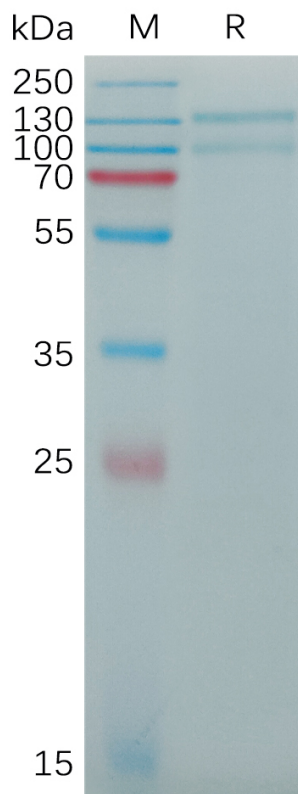


Figure 1. Human LDLR Protein, His Tag on SDS-PAGE under reducing condition.

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