

DESCRIPTION

Source Human embryonic kidney cell, HEK293-derived
Ala22-Arg788 (Asp193Ala), with a C-terminal 6-His tag
Accession # P01130

N-terminal Sequence Analysis Ala22

Predicted Molecular Mass 86 kDa

SPECIFICATIONS

SDS-PAGE 120-140 kDa, reducing conditions

Activity Measured in a competitive binding assay.
When human LDL is immobilized at 1 µg/mL (100 µL/well), Recombinant Human LDL R inhibits 50% binding of Biotinylated Recombinant Human LDL R (0.5 µg/mL) at the concentration range of 0.2-1.2 µg/mL.

Endotoxin Level <0.10 EU per 1 µg of the protein by the LAL method.

Purity >95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

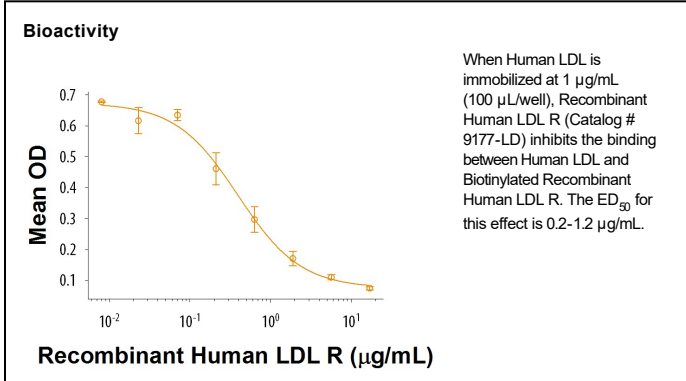
Reconstitution Reconstitute at 100 µg/mL in PBS.

Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA



BACKGROUND

The low density lipoprotein receptor (LDL R) is the founding member of the LDL R family of widely expressed cell surface scavenger receptors (1-5). Members of the family are endocytic receptors, but can also co-regulate adjacent cell-surface signaling molecules (3, 4). Many proteins in the LDL R family are cleaved by extracellular proteases to release soluble forms to the circulation, and many of these soluble forms are active (1, 6). Mature LDL R is a 120-160 kDa (depending on glycosylation) type I transmembrane glycoprotein that contains cysteine-rich complement-like repeats (class A LDL domains), calcium-binding EGF repeats, and β -propeller structures (class B LDL repeats) in the extracellular domain (ECD) (1-7). A membrane-proximal Ser/Thr-rich region shows extensive O-linked glycosylation (4, 8). A cytoplasmic NPxY motif links the LDL R to clathrin pits for endocytosis, and binds to select adaptor proteins (1, 4, 8). The human LDL R ECD shares 78%, 76%, 81% and 82% aa sequence identity with mouse, rat, bovine, and porcine LDL R, respectively. LDL R is constitutively and widely expressed. Its class A LDL domains near the N-terminus bind apoB and apoE, the apolipoproteins of low- and very low-density lipoproteins (LDL and VLDL), respectively (1, 2, 4, 9). Hepatocyte LDL R is responsible for endocytosis and clearing of most plasma LDL cholesterol (2, 9). At the low pH of the endocytic vesicle, it dissociates, allowing degradation of LDL and recycling of LDL R to the cell surface (1, 4). Lack of LDL R expression or function causes familial hypercholesterolemia (FH) (4, 9, 10). The protease PCSK9 (proprotein convertase subtilisin/kexin type 9) can also cause increased plasma cholesterol by promoting LDL R degradation rather than recycling to the cell surface (10-12). Soluble forms of approximately 140 kDa and 28 kDa are reported to be released by phorbol esters or interferons, respectively (6, 7).

References:

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