**DESCRIPTION**

**Source**
Mouse myeloma cell line, NS0-derived
Ala22-Arg788, with a C-terminal 6-His tag
Accession # Q59FQ1

**N-terminal Sequence Analysis**
Ala22 & Asp193

**Predicted Molecular Mass**
85.6 kDa & 66.8 kDa

**SPECIFICATIONS**

**SDS-PAGE**
125-140 kDa & 95-110 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 0.4-2 μg/mL.

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

**Purity**
>90%, by SDS-PAGE under reducing conditions and visualized by silver stain.

**Formulation**
Lyophilized from a 0.2 μm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

**Reconstitution**
Reconstitute at 100 μg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.

**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.

**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:**