Human CellExp™ VLDLR, human recombinant

CATALOG #:	7464-10	10 µg
	7464-50	50 µg

ALTERNATE NAMES: VLDLR, CARMQ1, CHRMQ1, FLJ35024, VLDLRCH, VLDL-R, very-low-density-lipoprotein receptor

SOURCE: HEK 293 cells (Gly 28 – Ser 797)

PURITY: ≥ 97% by SDS-PAGE gel

MOL. WEIGHT: This protein comprises 781 amino acids with polyhistidine tag at C-terminus and has a calculated MW of 86 kDa. The predicted N-terminus is Gly 28. DTT-reduced protein migrates as 150 & 180 kDa polypeptide in SDS-PAGE due to different glycosylation.

ENDOTOXIN LEVEL: <1 EU/µg by LAL method

FORM: Lyophilized

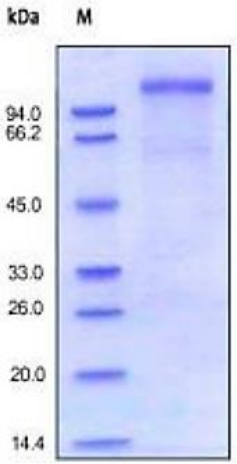
FORMULATION: Lyophilized from 0.22 µm filtered solution in PBS, pH 7.4. Normally Mannitol or Trehalose is added as protectants before lyophilization.

STORAGE CONDITIONS: Store at -20°C. After reconstitution, aliquot and store at -20°C and use within 3 months. Avoid repeated freezing and thawing cycles.

RECONSTITUTION: Centrifuge the vial prior to opening. Reconstitute in sterile PBS, pH 7.4 to a concentration of 50 µg/ml. Do not vortex. This solution can be stored at 2-8°C for up to 1 month. For extended storage, it is recommended to store at -20°C.

DESCRIPTION: The very-low-density-lipoprotein receptor (VLDL-R) is a lipoprotein receptor that shows considerable similarity to the low density-lipoprotein receptor. VLDL R is a 130 kDa type I transmembrane protein in the LDL receptor family that plays a significant role in lipid metabolism and in nervous system development and function .This receptor has been suggested to be important for the metabolism of apoprotein-E-containing triacylglycerol-rich lipoproteins, such as very-low-density lipoprotein (VLDL), beta-migrating VLDL and intermediate-density lipoprotein. It is also one of the receptors of reelin, an extracellular matrix protein which regulates the processes of neuronal migration and synaptic plasticity. In humans, the VLDL-R is encoded by the VLDLR gene. A rare neurological disorder first described in the 1970s under the name "disequilibrium syndrome" is now considered to be caused by the disruption of VLDLR gene. The disorder was renamed VLDLR-associated cerebellar hypoplasia (VLDLRCH) after a 2005 study. It is associated with parental consanguinity and found in secluded communities such as the Hutterites. VLDLRCH is one of the two known genetic disorders caused by a disruption of reelin signaling pathway, along with Norman-Roberts syndrome.

BIOLOGICAL ACTIVITY: Measured by its binding ability in a functional ELISA. When Recombinant Human Apolipoprotein E3 is immobilized at 1 µg/ml (100 µl/well), the concentration of Recombinant Human VLDLR that produces 50% of the optimal binding response is found to be approximately 0.03 - 0.15 µg/ml.



Human recombinant VLDLR

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FOR RESEARCH USE ONLY! Not to be used in humans.

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