

Product datasheet for **RC200006L3V**

LDL Receptor (LDLR) (NM_000527) Human Tagged ORF Clone Lentiviral Particle

Product data:

Product Type:	Lentiviral Particles
Product Name:	LDL Receptor (LDLR) (NM_000527) Human Tagged ORF Clone Lentiviral Particle
Symbol:	LDL Receptor
Synonyms:	FH; FHC; FHCL1; LDLCQ2
Mammalian Cell Selection:	Puromycin
Vector:	pLenti-C-Myc-DDK-P2A-Puro (PS100092)
Tag:	Myc-DDK
ACCN:	NM_000527
ORF Size:	2580 bp
ORF Nucleotide Sequence:	The ORF insert of this clone is exactly the same as(RC200006).
OTI Disclaimer:	The molecular sequence of this clone aligns with the gene accession number as a point of reference only. However, individual transcript sequences of the same gene can differ through naturally occurring variations (e.g. polymorphisms), each with its own valid existence. This clone is substantially in agreement with the reference, but a complete review of all prevailing variants is recommended prior to use. More info
OTI Annotation:	This clone was engineered to express the complete ORF with an expression tag. Expression varies depending on the nature of the gene.
RefSeq:	NM_000527.2 , NP_000518.1
RefSeq Size:	5175 bp
RefSeq ORF:	2583 bp
Locus ID:	3949
UniProt ID:	P01130
Cytogenetics:	19p13.2
Domains:	ldl_recept_b, EGF_CA, ldl_recept_a, EGF, EGF
Protein Families:	Druggable Genome, ES Cell Differentiation/IPS, Transmembrane



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Protein Pathways: Endocytosis

MW: 95.38 kDa

Gene Summary: 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]