

## DESCRIPTION

<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human LDL R in ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant mouse (rm) LDL R, recombinant human LRP-5, or rmlRP-6 is observed.
<b>Source</b>	Monoclonal Mouse IgG <sub>1</sub> Clone # 472413
<b>Purification</b>	Protein A or G purified from hybridoma culture supernatant
<b>Immunogen</b>	Chinese hamster ovary cell line CHO-derived recombinant human LDL R Ala22-Arg788 Accession # P01130
<b>Conjugate</b>	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 nm
<b>Formulation</b>	Supplied in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details.  *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

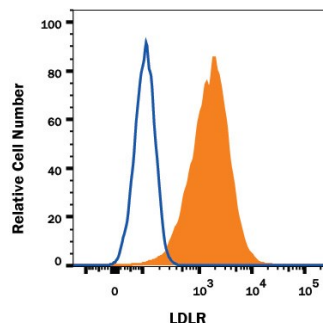
## APPLICATIONS

**Please Note:** Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Flow Cytometry</b>	0.25-1 µg/10 <sup>6</sup> cells	See Below

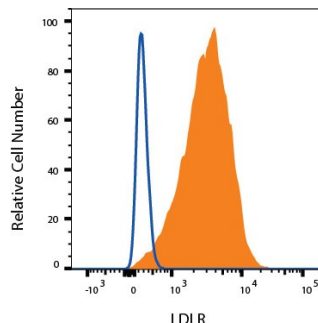
## DATA

### Flow Cytometry



**Detection of LDL R in HepG2 Human Cell Line by Flow Cytometry.** HepG2 human hepatocellular carcinoma cell line was stained with Mouse Anti-Human LDL R Alexa Fluor® 647-conjugated Monoclonal Antibody (Catalog # FAB2148R, filled histogram) or isotype control antibody (Catalog # IC002R, open histogram). View our protocol for [Staining Membrane-associated Proteins](#).

### Flow Cytometry



**Detection of LDLR in U-118-MG cells by Flow Cytometry** U-118-MG cells were stained with Mouse Anti-Human LDLR Alexa Fluor® 647-conjugated Monoclonal Antibody (Catalog # FAB2148R, filled histogram) or isotype control antibody (Catalog # IC002R, open histogram). View our protocol for [Staining Membrane-associated Proteins](#).

## PREPARATION AND STORAGE

<b>Shipping</b>	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<b>Protect from light. Do not freeze.</b> <ul style="list-style-type: none"> <li>12 months from date of receipt, 2 to 8 °C as supplied.</li> </ul>

## BACKGROUND

The low density lipoprotein receptor (LDL R) is the founding member of the LDL R family of scavenger receptors (1, 2). This family contains transmembrane molecules that are characterized by the presence of EGF repeats, complement-like repeats, and YWTD motifs that form  $\beta$ -propellers. Although members of the family were originally thought to be endocytic receptors, it is now clear that some members interact with adjacent cell-surface molecules, expanding their range of activities (2). Human LDL R is synthesized as an 860 amino acid (aa) precursor that contains a 21 aa signal sequence, a 767 aa extracellular region, a 22 aa transmembrane segment and a 50 aa cytoplasmic tail (3). The extracellular region is complex. It consists of seven N-terminal complement-like cysteine-rich repeats that bind ligand. Cysteine residues in this region participate in intrachain disulfide bonds. This region is followed by three EGF-like repeats with a  $\beta$ -propeller YWTD containing motif. The EGF-like repeats are responsible for ligand bonding and dissociation. Finally, there is a 50 aa membrane proximal Ser/Thr-rich region that serves as a carbohydrate attachment point (1, 3, 4). There is extensive O-linked and modest N-linked glycosylation. Thus the receptor's predicted molecular weight of 93 kDa is increased to a native molecular weight of 120-160 kDa (3, 4). Within the 50 aa cytoplasmic tail, there is an NPXY motif that links the receptor to clathrin pits (1). The extracellular region of human LDL R is 51% aa identical to the extracellular region of human VLDL R, and 79% aa identical to the extracellular region of mouse LDL R. LDL R is constitutively expressed and binds apoB of LDL and apoE of VLDL (5). It is responsible for clearing 70% of plasma LDL in liver (5). Mutations in the LDL R gene cause the autosomal dominant disorder, familial hypercholesterolemia (6).

## References:

1. Strickland, D.K. *et al.* (2002) Trends Endocrinol. Metab. **13**:66.
2. Nykjaer, A. and T.E. Willnow (2002) Trends Cell Biol. **12**:273.
3. Yamamoto, T. *et al.* (1984) Cell **39**:27.
4. Davis, C.G. *et al.* (1986) J. Biol. Chem. **261**:2828.
5. Defesche, J.C. (2004) Semin. Vasc. Med. **4**:5.
6. Varret, M. *et al.* (2008) Clin Genet. **73**:1.

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