

## DESCRIPTION

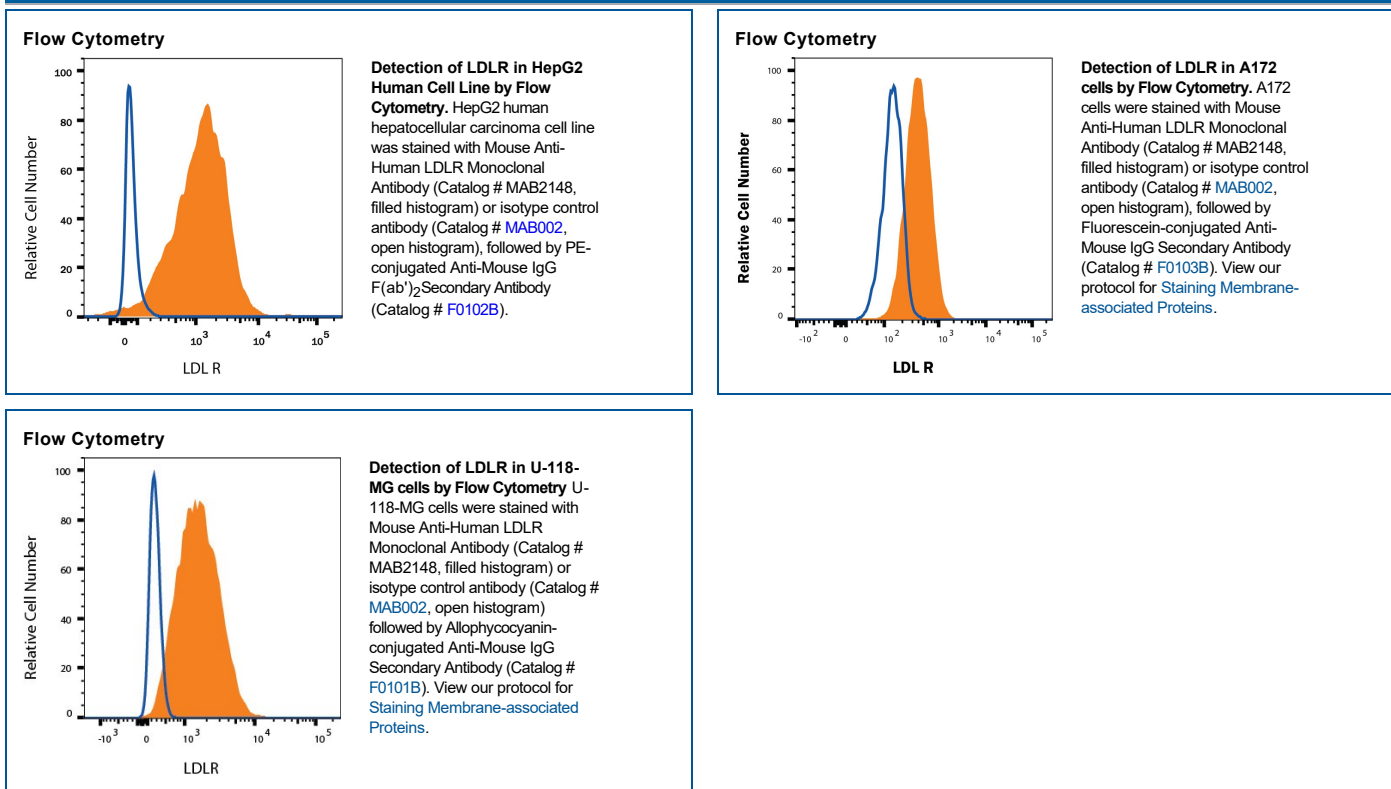
<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human LDLR in ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant mouse (rm) LDLR, recombinant human LRP-5, or rmlRP-6 is observed.
<b>Source</b>	Monoclonal Mouse IgG <sub>1</sub> Clone # 472413
<b>Purification</b>	Protein A or G purified from hybridoma culture supernatant
<b>Immunogen</b>	Chinese hamster ovary cell line CHO-derived recombinant human LDLR Ala22-Arg788 Accession # P01130
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose.

## 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>Western Blot</b>	1 µg/mL	Recombinant Human LDLR (Catalog # <a href="#">2148-LD</a> ) under non-reducing conditions only
<b>Flow Cytometry</b>	0.25 µg/10 <sup>6</sup> cells	See Below
<b>Immunoprecipitation</b>	25 µg/mL	Conditioned cell culture medium spiked with Recombinant Human LDLR (Catalog # <a href="#">2148-LD</a> ), <a href="#">see our available Western blot detection antibodies</a>
<b>Human LDLR Sandwich Immunoassay</b>		<b>Reagent</b>
<b>ELISA Capture</b>	2-8 µg/mL	Human LDLR Antibody (Catalog # <a href="#">MAB2148</a> )
<b>ELISA Detection</b>	0.1-0.4 µg/mL	Human LDLR Biotinylated Antibody (Catalog # <a href="#">BAF2148</a> )
<b>Standard</b>		Recombinant Human LDLR (Catalog # <a href="#">2148-LD</a> )
<b>CyTOF-ready</b>	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	

## DATA



#### PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.5 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<p><b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b></p> <ul style="list-style-type: none"> <li>• 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>• 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

#### BACKGROUND

The Low Density Lipoprotein Receptor (LDLR) is the founding member of the LDLR 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 LDLR 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 LDLR is 51% aa identical to the extracellular region of human VLDLR, and 79% aa identical to the extracellular region of mouse LDLR. LDLR 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 LDLR gene cause the autosomal dominant disorder, familial hypercholesterolemia (6).

#### References:

1. Strickland, D.K. *et al.* (2002) Trends Endocrinol. Metab. **13**:66.
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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.