

DESCRIPTION

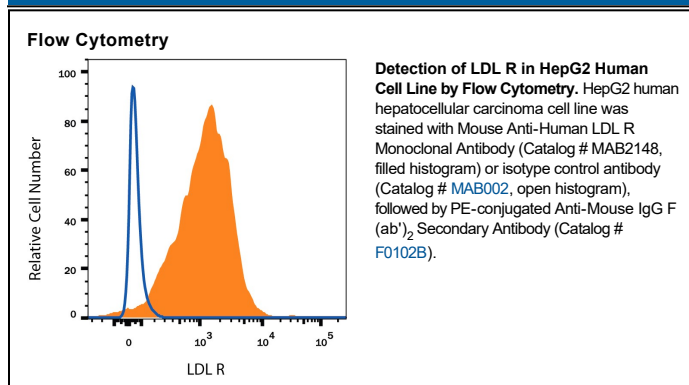
Species Reactivity	Human
Specificity	Detects human LDL R in ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant mouse (rm) LDL R, recombinant human LRP-5, or rmLRP-6 is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 472413
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Chinese hamster ovary cell line CHO-derived recombinant human LDL R Ala22-Arg788 Accession # P01130
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

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.

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

DATA



PREPARATION AND STORAGE

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