Human LDLR Alexa Fluor® 488-conjugated Antibody

Monoclonal Mouse IgG_1 Clone # 472413

Catalog Number: FAB2148G

100 Tests

DESCRIPTION

Species Reactivity	Human
Specificity	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.
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 LDLR Ala22-Arg788 Accession # P01130
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 nm
Formulation	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.

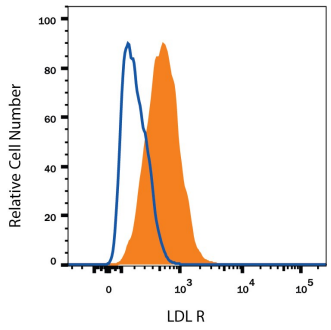
APPLICATIONS

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.*

	Recommended Concentration	Sample
Flow Cytometry	0.5 µg/10^6 cells	See Below

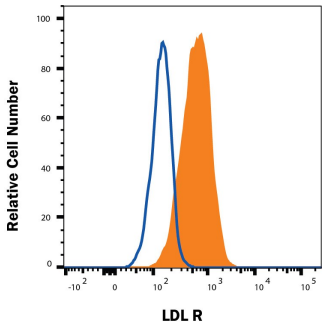
DATA

Flow Cytometry



Detection of LDLR in HepG2 Human Cell Line by Flow Cytometry. HepG2 human hepatocellular carcinoma cell line was stained with Mouse Anti-Human LDLR Alexa Fluor® 488-conjugated Monoclonal Antibody (Catalog # FAB2148G, filled histogram) or isotype control antibody (Catalog # IC002G, open histogram). View our protocol for Staining Membrane-associated Proteins.

Flow Cytometry



Detection of LDLR in A172 cells by Flow Cytometry. A172 cells were stained with Mouse Anti-Human LDLR Alexa Fluor® 488-conjugated Monoclonal Antibody (Catalog # FAB2148G, filled histogram) or isotype control antibody (Catalog # IC002G, open histogram). View our protocol for Staining Membrane-associated Proteins.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Protect from light. Do not freeze.** • 12 months from date of receipt, 2 to 8 °C as supplied.

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Human LDLR Alexa Fluor® 488-conjugated Antibody

Monoclonal Mouse IgG_1 Clone # 472413

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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 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 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 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 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.
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.

PRODUCT SPECIFIC NOTICES

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