# PRODUCT INFORMATION

## LDL Receptor Rabbit Monoclonal Antibody (Clone 004)

*Item No. 37076*

### Overview and Properties

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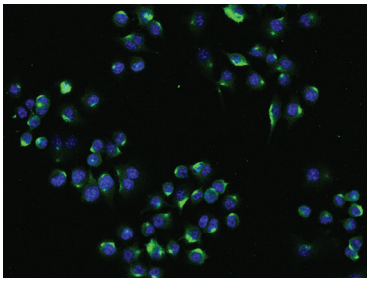
|---|---|
| **Contents:** | This vial contains 50 or 100 µl of protein A-affinity purified monoclonal antibody. |
| **Synonyms:** | LDLR, Low Density Lipoprotein Receptor |
| **Immunogen:** | Recombinant mouse LDLR |
| **Cross Reactivity:** | (+) LDLR |
| **Species Reactivity:** | (+) Mouse |
| **Form:** | Liquid |
| **Storage:** | -80°C (as supplied) |
| **Stability:** | ≥1 year |
| **Storage Buffer:** | 0.2 µm filtered solution in PBS |
| **Clone:** | 004 |
| **Host:** | Rabbit |
| **Isotype:** | IgG |
| **Applications:** | Immunocytochemistry (ICC) and immunofluorescence (IF). The recommended starting dilution is 1:20 for ICC and 1:100 for IF. Other applications were not tested, therefore optimal working concentration/dilution should be determined empirically. |

*Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.*

### Image



Immunofluorescent labeling of mouse LDLR in RAW 264.7 cells labeled with LDL Receptor Rabbit Monoclonal Antibody. Cells were fixed with 4% PFA, blocked with 10% serum, and incubated with LDL Receptor Rabbit Monoclonal Antibody (Clone 004) at 4°C overnight using a dilution of 1:60. Then cells were stained with the Alexa Fluor®488-conjugated Goat Anti-rabbit IgG secondary antibody (green) and counterstained with DAPI (blue).



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## Description

LDL receptor (LDLR) is a cell surface glycoprotein that scavenges LDL from the blood and regulates plasma LDL levels.[1] It is composed of an N-terminal signal sequence, a ligand-binding domain, an EGF precursor homology domain, an O-linked glycosylation domain, a transmembrane region, and a C-terminal cytoplasmic tail. LDLR is primarily expressed in the liver but is also found in the adrenal cortex.[2] It mediates the endocytosis of LDL by binding to apolipoprotein E (ApoE) or ApoB on the LDL surface, thereby supplying cholesterol to cells.[1] Protein levels of LDLR are decreased in HepG2 cells expressing proprotein convertase subtilisin kexin 9 (PCSK9).[3] Knockout of *Ldlr* increases plasma levels of cholesterol and triglycerides and induces the formation of atherosclerotic lesions in mice.[4] Mutations in *LDLR* are associated with familial hypercholesterolemia.[5] Cayman's LDL Receptor Rabbit Monoclonal Antibody (Clone 004) can be used for immunocytochemistry (ICC) and immunofluorescence (IF) applications.

## References


1. Goldstein, J.L., Brown, M.S., Anderson, R.G.W., *et al*. Receptor-mediated endocytosis: Concepts emerging from the LDL receptor system. *Annu. Rev. Cell Biol.* **1**, 1-39 (1985).
2. Rudling, M.J., Reihnér, E., Einarsson, K., *et al*. Low density lipoprotein receptor-binding activity in human tissues: Quantitative importance of hepatic receptors and evidence for regulation of their expression *in vivo*. *Proc. Natl. Acad. Sci. USA* **87(9)**, 3469-3473 (1990).
3. Benjannet, S., Rhainds, D., Essalmani, R., *et al*. NARC-1/PCSK9 and its natural mutants. Zymogen cleavage and effects on the low density lipoprotein (LDL) receptor and LDL cholesterol. *J. Biol. Chem.* **279(47)**, 48865-48875 (2004).
4. Praticò, D., Tillmann, C., Zhang, Z.B., *et al*. Acceleration of atherogenesis by COX-1-dependent prostanoid formation in low density lipoprotein receptor knockout mice. *Proc. Natl. Acad. Sci. USA* **98(6)**, 3358-3363 (2001).
5. Austin, M.A., Hutter, C.M., Zimmern, R.L., *et al*. Genetic causes of monogenic heterozgous familial hypercholesterolemia: A HuGE prevalence review. *Am. J. Epidemiol.* **160(5)**, 407-420 (2004).



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