



LDLR Monoclonal Antibody

BYmab-07098
lgG
Human;Rat;Mouse;Bovine
WB
LDLR
Low-density lipoprotein receptor (LDL receptor)
Synthesized peptide derived from human protein . at AA range: 540-620
LDLR Monoclonal Antibody detects endogenous levels of protein.
Liquid in PBS containing 50% glycerol, and 0.02% sodium azide.
Monoclonal, Mouse,IgG
The antibody was affinity-purified from mouse antiserum by affinity-chromatography using epitope-specific immunogen.
WB 1:500-2000
1 mg/ml
≥90%
-20°C/1 year
94kD
Cell membrane ; Single-pass type I membrane protein . Membrane, clathrin-coated pit . Golgi apparatus . Early endosome . Late endosome . Lysosome . Rapidly endocytosed upon ligand binding
Brain,Liver,Lymph,Plasma,
disease:Defects in LDLR are the cause of familial hypercholesterolemia (FH) [MIM:143890]; a common autosomal semi-dominant disease that affects about 1 in 500 individuals. The receptor defect impairs the catabolism of LDL, and the resultant elevation in plasma LDL-cholesterol promotes deposition of cholesterol in the skin (xanthelasma), tendons (xanthomas), and coronary arteries (atherosclerosis).,function:Binds LDL, the major cholesterol-carrying lipoprotein of plasma, and transports it into cells by endocytosis. In order to be internalized, the receptor-ligand complexes must first cluster into clathrin-coated pits. In case of HIV-1 infection, functions as a receptor for extracellular Tat in neurons, mediating its internalization in uninfected cells.,online information:LDLR mutation database,online information:The Singapore human mutation and polymorphism

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database,PTM:N- and O-glycosyla

Background	The low density lipoprotein receptor (LDLR) gene family consists of cell surface proteins involved in receptor-mediated endocytosis of specific ligands. Low density lipoprotein (LDL) is normally bound at the cell membrane and taken into the cell ending up in lysosomes where the protein is degraded and the cholesterol is made available for repression of microsomal enzyme 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase, the rate-limiting step in cholesterol synthesis. At the same time, a reciprocal stimulation of cholesterol ester synthesis takes place. Mutations in this gene cause the autosomal dominant disorder, familial hypercholesterolemia. Alternate splicing results in multiple transcript variants.[provided by RefSeq, Sep 2010],	
matters needing attention	Avoid repeated freezing and thawing!	
Usage suggestions	This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.	
Products Images		

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