



# APOB mouse mAb

<b>Catalog No</b>	YP-mAb-00782
<b>Isotype</b>	IgG
<b>Reactivity</b>	Human;Rat;Mouse;
<b>Applications</b>	WB
<b>Gene Name</b>	APOB
<b>Protein Name</b>	APOB
<b>Immunogen</b>	Synthesized peptide derived from human APOB
<b>Specificity</b>	This antibody detects endogenous levels of Human APOB
<b>Formulation</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
<b>Source</b>	Monoclonal, Mouse,IgG
<b>Purification</b>	The antibody was affinity-purified from mouse antiserum by affinity-chromatography using epitope-specific immunogen.
<b>Dilution</b>	WB 1:500-1:2000
<b>Concentration</b>	1 mg/ml
<b>Purity</b>	≥90%
<b>Storage Stability</b>	-20°C/1 year
<b>Synonyms</b>	Apolipoprotein B-100 (Apo B-100) [Cleaved into: Apolipoprotein B-48 (Apo B-48)]
<b>Observed Band</b>	
<b>Cell Pathway</b>	Cytoplasm . Secreted . Lipid droplet .
<b>Tissue Specificity</b>	
<b>Function</b>	disease:Defects in APOB are a cause of familial hypobetalipoproteinemia (FHBL) [MIM:107730]. FHBL is a genetically heterogeneous autosomal co-dominant disorder, associated with reduced plasma concentrations of apoB, LDL and VLDL. Heterozygotes for FHBL are usually asymptomatic with LDL cholesterol and apoB-100 concentrations less than 50% of those in normal plasma. Homozygotes have extremely low plasma LDL cholesterol and apoB-100 concentrations, and clinical presentation may vary from no symptoms to severe gastrointestinal and neurological dysfunction similar to abetalipoproteinemia [MIM:200100].,disease:Defects in APOB are a cause of familial ligand-defective apolipoprotein B-100 (FDB) [MIM:144010]. FDB is a dominantly inherited disorder of lipoprotein metabolism leading to hypercholesterolemia and increased proneness to coronary artery disease (CAD). The plasma cholesterol levels are
<b>Background</b>	This gene product is the main apolipoprotein of chylomicrons and low density lipoproteins. It occurs in plasma as two main isoforms, apoB-48 and apoB-100: the former is synthesized exclusively in the gut and the latter in the liver. The



intestinal and the hepatic forms of apoB are encoded by a single gene from a single, very long mRNA. The two isoforms share a common N-terminal sequence. The shorter apoB-48 protein is produced after RNA editing of the apoB-100 transcript at residue 2180 (CAA->UAA), resulting in the creation of a stop codon, and early translation termination. Mutations in this gene or its regulatory region cause hypobetalipoproteinemia, normotriglyceridemic hypobetalipoproteinemia, and hypercholesterolemia due to ligand-defective apoB, diseases affecting plasma cholesterol and apoB levels. [provided by RefSeq, Jul 2008],

**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