

## Lamin B2 mouse mAb

<b>Catalog No :</b>	YM1521
<b>Reactivity :</b>	Human;Mouse
<b>Applications :</b>	WB
<b>Target :</b>	Lamin B2
<b>Fields :</b>	>>Apoptosis
<b>Gene Name :</b>	Lamin B2
<b>Human Gene Id :</b>	84823
<b>Human Swiss Prot No :</b>	Q03252
<b>Mouse Swiss Prot No :</b>	P21619
<b>Immunogen :</b>	Recombinant human Lamin B2 protein.
<b>Specificity :</b>	This antibody detects endogenous levels of Lamin B2 and does not cross-react with related proteins.
<b>Formulation :</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
<b>Source :</b>	Monoclonal, Mouse
<b>Dilution :</b>	wb dilution 1:500
<b>Purification :</b>	The antibody was affinity-purified from mouse ascites by affinity-chromatography using epitope-specific immunogen.
<b>Concentration :</b>	1 mg/ml
<b>Storage Stability :</b>	-15°C to -25°C/1 year (Do not lower than -25°C)
<b>Observed Band :</b>	68kD

## Background :

lamin B2(LMNB2) Homo sapiens This gene encodes a B type nuclear lamin. The nuclear lamina consists of a two-dimensional matrix of proteins located next to the inner nuclear membrane. The lamin family of proteins make up the matrix and are highly conserved in evolution. During mitosis, the lamina matrix is reversibly disassembled as the lamin proteins are phosphorylated. Lamin proteins are thought to be involved in nuclear stability, chromatin structure and gene expression. Vertebrate lamins consist of two types, A and B. Mutations in this gene are associated with acquired partial lipodystrophy. [provided by RefSeq, May 2012],

## Function :

disease:Defects in LMNB2 are a cause of partial acquired lipodystrophy (APL) [MIM:608709]; also called Barraquer-Simons syndrome. APL is a rare childhood disease characterized by loss of subcutaneous fat from the face and trunk. Fat deposition on the pelvic girdle and lower limbs is normal or excessive. Most frequently, onset between 5 and 15 years of age. Most affected subjects are females and some show no other abnormality, but many develop glomerulonephritis, diabetes mellitus, hyperlipidaemia, and complement deficiency. Mental retardation in some cases. APL is a sporadic disorder of unknown aetiology.,function:Lamins are components of the nuclear lamina, a fibrous layer on the nucleoplasmic side of the inner nuclear membrane, which is thought to provide a framework for the nuclear envelope and may also interact with chromatin.,miscellaneous:The structural integrity of the lamina is s

## Subcellular Location :

Nucleus lamina .

## Expression :

Epithelium,Fetal brain cortex,Muscle,

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