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# Lamin A/C Polyclonal Antibody

YP-Ab-00428
IgG
Human;Mouse;Rat
WB;IHC;IF;ELISA
LMNA
Prelamin-A/C
The antiserum was produced against synthesized peptide derived from human Lamin A/C. AA range:361-410
Lamin A/C Polyclonal Antibody detects endogenous levels of Lamin A/C protein.
Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Polyclonal, Rabbit,IgG
The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/20000. Not yet tested in other applications.
1 mg/ml
≥90%
-20°C/1 year
LMNA; LMN1; Prelamin-A/C
74+65kD
Nucleus . Nucleus envelope . Nucleus lamina. Nucleus, nucleoplasm. Nucleus matrix . Farnesylation of prelamin-A/C facilitates nuclear envelope targeting and subsequent cleavage by ZMPSTE24/FACE1 to remove the farnesyl group produces mature lamin-A/C, which can then be inserted into the nuclear lamina. EMD is required for proper localization of non-farnesylated prelamin-A/C.; [Isoform C]: Nucleus speckle .
In the arteries, prelamin-A/C accumulation is not observed in young healthy vessels but is prevalent in medial vascular smooth muscle cells (VSMCs) from aged individuals and in atherosclerotic lesions, where it often colocalizes with senescent and degenerate VSMCs. Prelamin-A/C expression increases with age and disease. In normal aging, the accumulation of prelamin-A/C is caused in part by the down-regulation of ZMPSTE24/FACE1 in response to oxidative stress.
disease:Defects in LMNA are a cause of Emery-Dreifuss muscular dystrophy type 2 (EDMD2) [MIM:181350]. EDMD2 is an autosomal dominant disorder characterized by slowly progressive muscle wasting and weakness, early contractures of the elbows Achilles tendons and spine, and cardiomyopathy associated with cardiac conduction defects.,disease:Defects in LMNA are a cause



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	of Emery-Dreifuss muscular dystrophy type 3 (EDMD3) [MIM:604929]. EDMD3 is an autosomal recessive disorder characterized by early contractures, muscle wasting and weakness and cardiomyopathy.,disease:Defects in LMNA are a cause of familial partial lipodystrophy type 2 (FPLD2) [MIM:151660]; also known as familial partial lipodystrophy Dunnigan type. FPLD2 is an autosomal dominant disorder characterized by marked loss of subcutaneous adipose tissue from the extremities and trunk but by excess fat deposition in the head and neck.
Background	lamin A/C(LMNA) Homo sapiens 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. Alternative splicing results in multiple transcript variants. Mutations in this gene lead to several diseases: Emery-Dreifuss muscular dystrophy, familial partial lipodystrophy, limb girdle muscular dystrophy, dilated cardiomyopathy, Charcot-Marie-Tooth disease, and Hutchinson-Gilford progeria syndrome. [provided by RefSeq, Apr 2012],
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.

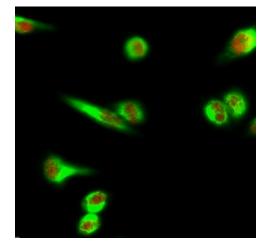


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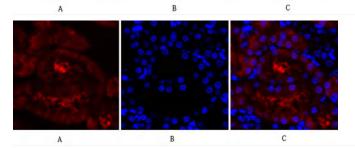


#### **Products Images**

Immunofluorescence analysis of Hela cell. 1,Lamin A/C Polyclonal Antibody(red) was diluted at 1:200(4° overnight). Galectin-3 Monoclonal Antibody(6G2)(green) was diluted at 1:200(4° overnight). 2, Goat Anti Rabbit Alexa Fluor 594 Catalog:RS3611 was diluted at 1:1000(room temperature, 50min). Goat Anti Mouse Alexa Fluor 488 Catalog:RS3208 was diluted at 1:1000(room temperature, 50min).



BCImmunofluorescence analysis of human-liver tissue.<br/>1,Lamin A/C Polyclonal Antibody(red) was diluted at<br/>1:200(4°C,overnight). 2, Cy3 labled Secondary<br/>antibody was diluted at 1:300(room temperature,<br/>50min).3, Picture B: DAPI(blue) 10min. Picture<br/>A:Target. Picture B: DAPI. Picture C: merge of A+BBcImmunofluorescence analysis of human-liver tissue.<br/>1,Lamin A/C Polyclonal Antibody(red) was diluted at<br/>1:200(4°C,overnight). 2, Cy3 labled Secondary<br/>antibody was diluted at<br/>1:200(4°C,overnight). 2, Cy3 labled Secondary<br/>antibody was diluted at 1:200(4°C,overnight). 2, Cy3 labled Secondary<br/>antibody was diluted at 1:300(room temperature,<br/>50min).3, Picture B: DAPI(blue) 10min. Picture<br/>A:Target. Picture B: DAPI(blue) 10min. Picture<br/>A:Target. Picture B: DAPI. Picture C: merge of A+B



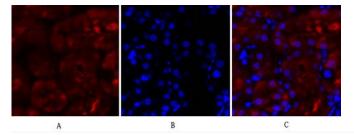
Immunofluorescence analysis of rat-kidney tissue. 1,Lamin A/C Polyclonal Antibody(red) was diluted at 1:200(4°C,overnight). 2, Cy3 labled Secondary antibody was diluted at 1:300(room temperature, 50min).3, Picture B: DAPI(blue) 10min. Picture A:Target. Picture B: DAPI. Picture C: merge of A+B



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Immunofluorescence analysis of rat-kidney tissue. 1,Lamin A/C Polyclonal Antibody(red) was diluted at 1:200(4°C,overnight). 2, Cy3 labled Secondary antibody was diluted at 1:300(room temperature, 50min).3, Picture B: DAPI(blue) 10min. Picture A:Target. Picture B: DAPI. Picture C: merge of A+B



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