

🔇 Tel: 400-999-8863 📼 Emall:Upingbio.163.com

Website: www.upingBio.com

Cleaved-Lamin A (D230) Polyclonal Antibody

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		
Reactivity Human;Mouse;Rat Applications WB;IHC;IF;ELISA Gene Name LMNA Protein Name Prelamin-A/C Immunogen The antiserum was produced against synthesized peptide derived from human Lamin A. AA range:181-230 Specificity Cleaved-Lamin A (D230) Polyclonal Antibody detects endogenous levels of fragment of activated Lamin A protein resulting from cleavage adjacent to D230. Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide. Source Polyclonal, Rabbit,IgG Purification The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. Dilution WB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/1000 IF 1:50-200 Concentration 1 mg/ml Purity ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway 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; Ilsoform C]; Nucleus speckle. Tissue Specificity In the arteries, prelamin-A/C accumulation of prelamin-A/C; Ilsoform C]; Nucleus speckle. <td< th=""><th>Catalog No</th><td>YP-Ab-00025</td></td<>	Catalog No	YP-Ab-00025
Applications WB;IHC;IF;ELISA Gene Name LMNA Protein Name Prelamin-A/C Immunogen The antiserum was produced against synthesized peptide derived from human Lamin A. AA range:181-230 Specificity Cleaved-Lamin A (D230) Polyclonal Antibody detects endogenous levels of fragment of activated Lamin A protein resulting from cleavage adjacent to D230. Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide. Source Polyclonal, Rabbit,IgG Purification The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. Dilution WB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/10000 IF 1:50-200 Concentration 1 mg/ml Purify ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway Nucleus, Nucleus envelope , Nucleus lamina, Nucleus, nucleoplasm. Nucleus Insofter proper localization of non-farnesylated prelamin-A/C; ifsoftom C1; Nucleus speckle Tissue Specificity In the arteries, prelamin-A/C accumulation of prelamin-A/C, sprease in normal aging, the accumulation of prelamin-A/C; ifsoftom C1; Nucleus speckle Tissue Specificity In th	Isotype	lgG
Gene Name LMNA Protein Name Prelamin-A/C Immunogen The antiserum was produced against synthesized peptide derived from human Lamin A. AA range: 181-230 Specificity Cleaved-Lamin A (D230) Polyclonal Antibody detects endogenous levels of fragment of activated Lamin A protein resulting from cleavage adjacent to D230. Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide. Source Polyclonal, Rabbit,IgG Purification The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. Dilution WB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/10000 IF 1:50-200 Concentration 1 mg/ml Purity ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Cell Pathway Nucleus envelope . Nucleus lamina. Nucleus, nucleoplasm. Nucleus matrix. Farmesylation of prelamin-A/C facilitates nuclear envelope targeting and subsequent cleavage by ZMPSTEEL/FACE1 to remove the farmesyl group produces mature lamin-A/C, which can then be inserted into the nuclear lamina. EMD is required for proper localization of non-farmesylated prelamin-A/C, illosoff mor aged individuals and in atherosclerotic lesions, where it ofen colocalizes with sensence it in medial vascular models (VSMCS) from aged individue sind in atherosclerotic lesions. Moreet oxiditive stress. The advind usease. In normal aging,	Reactivity	Human;Mouse;Rat
Protein Name Prelamin-A/C Immunogen The antiserum was produced against synthesized peptide derived from human Lamin A. AA range:181-230 Specificity Cleaved-Lamin A (D230) Polycolonal Antibody detects endogenous levels of fragment of activated Lamin A protein resulting from cleavage adjacent to D230. Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide. Source Polyclonal, Rabbit,IgG Purification The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. Dilution WB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/10000 IF 1:50-200 Concentration 1 mg/ml Purity ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway 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 accumulation is not observed in young healthy vessels but is prevalent in medial vascular smooth muscle (SMCS) from aged individuals and in atherosclerotic lesions, where it often colocalizes with seenscent in medial vascular smooth muscle (SMCS) from aged individuals and in atherosclerotic lesion, where it often colocalizes with seenscent and degenerate VSMRCS.	Applications	WB;IHC;IF;ELISA
Immunogen The antiserum was produced against synthesized peptide derived from human Lamin A. AA range:181-230 Specificity Cleaved-Lamin A (D230) Polyclonal Antibody detects endogenous levels of fragment of activated Lamin A protein resulting from cleavage adjacent to D230. Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide. Source Polyclonal, Rabbit,IgG Purification The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. Dilution WB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/10000 IF 1:50-200 Concentration 1 mg/ml Purity ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway Nucleus envelope. Nucleus lamina. Nucleus, nucleoplasm. Nucleus matrix . Farnesylation of prelamin-A/C facilitates nuclear envelope targeting and subsequent cleavage by ZMPS TE24/FACE 1 to remove the farmesyl gloup produces mature lamin-A/C, which can then be inserted into the nuclear lamina. EMD is required for proper localization of non-farmesylated prelamin-A/C ; [Isoform C]: Nucleus speckle . Tissue Specificity 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 a theresclerotil esions, where it often col	Gene Name	LMNA
Lamin A. AA range:181-230 Specificity Cleaved-Lamin A (D230) Polyclonal Antibody detects endogenous levels of fragment of activated Lamin A protein resulting from cleavage adjacent to D230. Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide. Source Polyclonal, Rabbit,IgG Purification The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. Dilution WB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/10000 IF 1:50-200 Concentration 1 mg/ml Purity ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway 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. Tissue Specificity 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 offen colocalizes with senerest 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. Function	Protein Name	Prelamin-A/C
FormulationLiquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.SourcePolyclonal, Rabbit,IgGPurificationThe antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.DilutionWB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/10000 IF 1:50-200Concentration1 mg/mlPurity≥90%Storage Stability-20°C/1 yearSynonymsLMNA; LMN1; Prelamin-A/CObserved Band28+75kDCell PathwayNucleus . 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; filsoform C]: Nucleus speckle .Tissue SpecificityIn 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 attherosclerotic 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 expression increases with age and dividuals and in attherosclerotic lesions, where it often colocalizesFunctiondisease-Defects in LMNA are a cause of Emery-Dreifus 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 condu	Immunogen	
Source Polyclonal, Rabbit,IgG Purification The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. Dilution WB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/10000 IF 1:50-200 Concentration 1 mg/ml Purity ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway 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 renewed the farnesyl group produces mature lamina. EMD is required for proper localization of non-farnesylated prelamin-A/C.; [Isoform C]: Nucleus speckle. Tissue Specificity 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 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. Function disease:Defects in LMNA are a cause of Emry-Dreifuss muscular dystrophy type 2 (EDMD2) [MIN: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,		
Purification The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen. Dilution WB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/10000 IF 1:50-200 Concentration 1 mg/ml Purity ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway 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 . Tissue Specificity 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 is caused in part by the down-regulation of ZMPSTE24/FACE1 in response to oxidative stress. Function 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 causes		
affinity-chromatography using epitope-specific immunogen. Dilution WB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/10000 IF 1:50-200 Concentration 1 mg/ml Purity ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway 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 . Tissue Specificity 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. Function 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	Source	Folycional, Rabbit,198
Concentration 1 mg/ml Purity ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway 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 . Tissue Specificity 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 is caused in part by the down-regulation of ZMPSTE24/FACE1 in response to oxidative stress. Function 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. Function 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	Purification	
Purity ≥90% Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway 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 . Tissue Specificity 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 attherosclerotic lesions, where it offen 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. Function 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 defectsdisease:Defects in LMNA are a cause	Dilution	WB: 1/500 - 1/2000. IHC: 1/100 - 1/300. ELISA: 1/10000 IF 1:50-200
Storage Stability -20°C/1 year Synonyms LMNA; LMN1; Prelamin-A/C Observed Band 28+75kD Cell Pathway 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 . Tissue Specificity 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. Function 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	Concentration	1 mg/ml
SynonymsLMNA; LMN1; Prelamin-A/CObserved Band28+75kDCell PathwayNucleus . 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 .Tissue SpecificityIn 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 as ensecent 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.Functiondisease: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	Purity	≥90%
Observed Band 28+75kD Cell Pathway 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 . Tissue Specificity 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 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. Function 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	Storage Stability	-20°C/1 year
Cell PathwayNucleus . 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 .Tissue SpecificityIn 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.Functiondisease: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	Synonyms	LMNA; LMN1; Prelamin-A/C
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 .Tissue SpecificityIn 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.Functiondisease: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	Observed Band	28+75kD
 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. Function 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 	Cell Pathway	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.;
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	Tissue Specificity	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
	Function	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



UpingBio technology Co.,Ltd

🔇 Tel: 400-999-8863 📼 Emall:Upingbio.163.com

Website: www.upingBio.com 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], Avoid repeated freezing and thawing! matters needing attention This product can be used in immunological reaction related experiments. For Usage suggestions more information, please consult technical personnel.



UpingBio technology Co.,Ltd

🔇 Tel: 400-999-8863 📼 Email:Upingbio.163.com

Ø Website: www.upingBio.com

Products Images



-- 26

-- 19 (kD)



UpingBio technology Co.,Ltd

🔇 Tel: 400-999-8863 📼 Email:Upingbio.163.com

Website: www.upingBio.com