

Product datasheet

Lamin B Receptor/LBR peptide ab45944

Overview

Product name	Lamin B Receptor/LBR peptide
Description	Mouse Lamin B Receptor/LBR peptide

Description

Nature	Synthetic
Amino Acid Sequence	
Species	Mouse

Specifications

Our [Abpromise guarantee](#) covers the use of **ab45944** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Form	Liquid
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Additional notes	<ul style="list-style-type: none">- First try to dissolve a small amount of peptide in either water or buffer. The more charged residues on a peptide, the more soluble it is in aqueous solutions.- If the peptide doesn't dissolve try an organic solvent e.g. DMSO, then dilute using water or buffer.- Consider that any solvent used must be compatible with your assay. If a peptide does not dissolve and you need to recover it, lyophilise to remove the solvent.- Gentle warming and sonication can effectively aid peptide solubilisation. If the solution is cloudy or has gelled the peptide may be in suspension rather than solubilised.- Peptides containing cysteine are easily oxidised, so should be prepared in solution just prior to use.
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This product was previously labelled as Lamin B Receptor

Preparation and Storage

Stability and Storage	Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles. Information available upon request.
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General Info

Function	Anchors the lamina and the heterochromatin to the inner nuclear membrane.
Involvement in disease	<p>Defects in LBR are a cause of Pelger-Huet anomaly (PHA) [MIM:169400]. PHA is an autosomal dominant inherited abnormality of neutrophils, characterized by reduced nuclear segmentation and an apparently looser chromatin structure. Heterozygotes show hypolobulated neutrophil nuclei with coarse chromatin. Presumed homozygous individuals have ovoid neutrophil nuclei, as well as varying degrees of developmental delay, epilepsy, and skeletal abnormalities.</p> <p>Defects in LBR are the cause of hydrops-ectopic calcification-moth-eaten skeletal dysplasia (HEM) [MIM:215140]; also known as Greenberg skeletal dysplasia. HEM is a rare autosomal recessive chondrodystrophy characterized by early in utero lethality and, therefore, considered to be nonviable. Affected fetuses typically present with fetal hydrops, short-limbed dwarfism, and a marked disorganization of chondro-osseous calcification and may present with polydactyly and additional nonskeletal malformations.</p> <p>Defects in LBR may be a cause of Reynolds syndrome (REYNS) [MIM:613471]. It is a syndrome specifically associating limited cutaneous systemic sclerosis and primary biliary cirrhosis. It is characterized by liver disease, telangiectasia, abrupt onset of digital paleness or cyanosis in response to cold exposure or stress (Raynaud phenomenon), and variable features of scleroderma. The liver disease is characterized by pruritis, jaundice, hepatomegaly, increased serum alkaline phosphatase and positive serum mitochondrial autoantibodies, all consistent with primary biliary cirrhosis.</p>
Sequence similarities	Belongs to the ERG4/ERG24 family.
Post-translational modifications	Phosphorylated by CDK1 protein kinase in mitosis when the inner nuclear membrane breaks down into vesicles that dissociate from the lamina and the chromatin. It is phosphorylated by different protein kinases in interphase when the membrane is associated with these structures. Phosphorylation of LBR and HP1 proteins may be responsible for some of the alterations in chromatin organization and nuclear structure which occur at various times during the cell cycle.
Cellular localization	Nucleus inner membrane.

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