abcam

Product datasheet

Mouse Lamin B1 peptide ab16375

3 References 1 Image

Description

Product name	Mouse Lamin B1 peptide
Purity	> 70 % HPLC. 70 - 90% by HPLC
Accession	**P20700**
Animal free	No
Nature	Synthetic
Species	Mouse

Specifications

Our **Abpromise guarantee** covers the use of **ab16375** in the following tested applications.

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

Applications	Blocking - Blocking peptide for Anti-Lamin B1 antibody - Nuclear Envelope Marker (**ab16048**)
Form	Lyophilized
Additional notes	*- 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.*

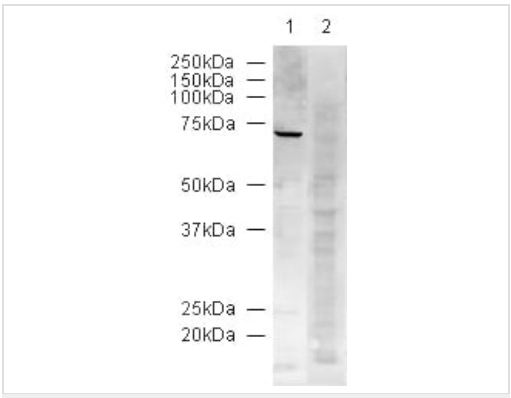
Preparation and Storage

Stability and Storage	Shipped at 4°C. Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle. Information available upon request.
Reconstitution	Reconstitute with 100 ul of deionised water (or equivalent).

General Info

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.
Involvement in disease	Defects in LMNB1 are the cause of leukodystrophy demyelinating autosomal dominant adult-onset (ADLD) [MIM:169500]. ADLD is a slowly progressive and fatal demyelinating leukodystrophy, presenting in the fourth or fifth decade of life. Clinically characterized by early autonomic abnormalities, pyramidal and cerebellar dysfunction, and symmetric demyelination of the CNS. It differs from multiple sclerosis and other demyelinating disorders in that neuropathology shows preservation of oligodendroglia in the presence of subtotal demyelination and lack of astrogliosis.
Sequence similarities	Belongs to the intermediate filament family.
Post-translational modifications	B-type lamins undergo a series of modifications, such as farnesylation and phosphorylation. Increased phosphorylation of the lamins occurs before envelope disintegration and probably plays a role in regulating lamin associations.
Cellular localization	Nucleus inner membrane.

Images



Western blot - Mouse Lamin B1 peptide (ab16375)

All lanes : Anti-Lamin B1 antibody - Nuclear Envelope Marker (**ab16048**) at 1/1000 dilution

Lane 1 : HeLa (Human cervix adenocarcinoma epithelial cell) whole cell lysate
Lane 2 : HeLa (Human cervix adenocarcinoma epithelial cell) whole cell lysate with Mouse Lamin B1 peptide (ab16375) at 1 µg/ml

Lysates/proteins at 20 µg per lane.

Secondary
All lanes : Alexa fluor goat polyclonal to Rabbit IgG at 1/10000 dilution

Performed under reducing conditions.

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