SANTA CRUZ BIOTECHNOLOGY, INC.

Lamin A (C-20): sc-6214

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

A unique family of cysteine proteases has been described that differs in sequence, structure and substrate specificity from any previously described protease family. This family, termed Ced-3/ICE, is comprised of ICE, CPP32, ICH-1/Nedd-2, Tx, Mch2, Mch3 (ICE-LAP3 or CMH-1), Mch4 and ICE-LAP6. Ced-3/ICE family members function as key components of the apoptotic machinery and act to destroy specific target proteins which are critical to cellular longevity. Nuclear lamins are critical to maintaining the integrity of the nuclear envelope and cellular morphology. The nuclear Lamin A is cleaved by Mch2, but not CPP32. Nuclear Lamin B is fragmented as a consequence of apoptosis by an unidentified member of the ICE family. Lamin C is a splice variant of Lamin A, differing only at the carboxy-terminus. Lamins A and C are identical for the first 566 amino acids, with Lamin C differing only in 6 unique carboxy-terminal amino acids.

CHROMOSOMAL LOCATION

Genetic locus: LMNA (human) mapping to 1q22; Lmna (mouse) mapping to 3 F1.

SOURCE

Lamin A (C-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of Lamin A of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-6214 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

Lamin A (C-20) is recommended for detection of Lamin A of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000); non cross-reactive with Lamin C.

Lamin A (C-20) is also recommended for detection of Lamin A in additional species, including equine, canine and porcine.

Suitable for use as control antibody for Lamin A/C siRNA (h): sc-35776, Lamin A/C siRNA (m): sc-29385, Lamin A/C shRNA Plasmid (h): sc-35776-SH, Lamin A/C shRNA Plasmid (m): sc-29385-SH, Lamin A/C shRNA (h) Lentiviral Particles: sc-35776-V and Lamin A/C shRNA (m) Lentiviral Particles: sc-29385-V.

Molecular Weight of Lamin A: 69 kDa.

Positive Controls: CCD-1064Sk cell lysate: sc-2263, Hs68 cell lysate: sc-2230 or C32 whole cell lysate: sc-2205.

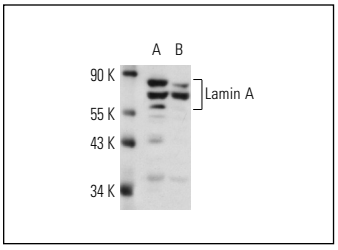
RESEARCH USE

For research use only, not for use in diagnostic procedures.

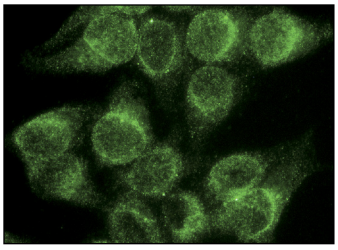
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

DATA



Lamin A (C-20): sc-6214. Western blot analysis of Lamin A expression in Hs68 (**A**) and C32 (**B**) whole cell lysates.



Lamin A (C-20): sc-6214. Immunofluorescence staining of methanol-fixed HeLa cells showing nuclear envelope localization.

SELECT PRODUCT CITATIONS

1. Pendás, A.M., et al. 2002. Defective prelamin A processing and muscular and adipocyte alterations in Zmpste24 metalloproteinase-deficient mice. Nat. Genet. 31: 94-99.
2. Horie, R., et al. 2002. Cytoplasmic aggregation of TRAF2 and TRAF5 proteins in the Hodgkin-Reed-Sternberg cells. Am. J. Pathol. 160: 1647-1654.
3. Bergo, M.O., et al. 2002. Zmpste24 deficiency in mice causes spontaneous bone fractures, muscle weakness, and a prelamin A processing defect. Proc. Natl. Acad. Sci. USA 99: 13049-13054.
4. Avnet, S., et al. 2011. Osteoblasts from a mandibuloacral dysplasia patient induce human blood precursors to differentiate into active osteoclasts. Biochim. Biophys. Acta 1812: 711-718.
5. Cenni, V., et al. 2011. Autophagic degradation of farnesylated prelamin A as a therapeutic approach to lamin-linked progeria. Eur. J. Histochem. 55: e36.
6. Capanni, C., et al. 2012. Familial partial lipodystrophy, mandibuloacral dysplasia and restrictive dermopathy feature barrier-to-autointegration factor (BAF) nuclear redistribution. Cell Cycle 11: 3568-3577.
7. Perrin, S., et al. 2012. HIV protease inhibitors do not cause the accumulation of prelamin A in PBMCs from patients receiving first line therapy: the ANRS EP45 "aging" study. PLoS ONE 7: e53035.
8. Bertrand, A.T., et al. 2012. DelK32-lamin A/C has abnormal location and induces incomplete tissue maturation and severe metabolic defects leading to premature death. Hum. Mol. Genet. 21: 1037-1048.