SANTA CRUZ BIOTECHNOLOGY, INC.

B-Myb (C-20): sc-725

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The highly leukemogenic avian retrovirus E26 contains two oncogenes, v-Myb and v-Ets, which are expressed together as a fusion protein. The cellular homolog of v-Myb, designated c-Myb, encodes a transcription factor. Deletion or disruption of a negative regulatory domain mapping within the carboxy-terminal domain of c-Myb results in enhanced transactivating capacity and in parallel, leads to activation of its ability to transform hemopoietic cells. c-Myb is expressed preferentially, but not exclusively, in immature hemopoietic cells and its expression decreases as cells differentiate. A second member of the Myb proto-oncogene family, B-Myb, encodes a second sequence-specific DNA binding protein. B-Myb RNA levels are low or undetectable in quiescent cells but increase at the G_1/S-phase transition following mitogenic stimulation. Studies suggest that B-Myb expression rescues cells from p53-induced G_1 arrest mediated by p21.

CHROMOSOMAL LOCATION

Genetic locus: MYBL2 (human) mapping to 20q13.12; Mybl2 (mouse) mapping to 2 H2.

SOURCE

B-Myb (C-20) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of B-Myb of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin. Also available as TransCruz reagent for Gel Supershift and ChIP applications, sc-725 X, 200 µg/0.1 ml.

Blocking peptide available for competition studies, sc-725 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

B-Myb (C-20) is recommended for detection of B-Myb of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:50-1:500), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:25, dilution range 1:25-1:250) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000). B-Myb (C-20) is also recommended for detection of B-Myb in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for B-Myb siRNA (h): sc-43523, B-Myb siRNA (m): sc-43524, B-Myb shRNA Plasmid (h): sc-43523-SH, B-Myb shRNA Plasmid (m): sc-43524-SH, B-Myb shRNA (h) Lentiviral Particles: sc-43523-V and B-Myb shRNA (m) Lentiviral Particles: sc-43524-V.

B-Myb (C-20) X TransCruz antibody is recommended for Gel Supershift and ChIP applications.

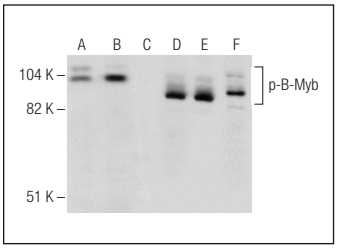
Molecular Weight of B-Myb: 110 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200.

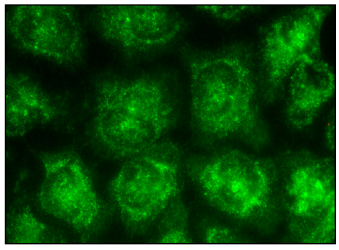
STORAGE

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

DATA



Western blot analysis of B-Myb phosphorylation in untreated (**A**,**D**), PMA treated (**B**,**E**) and PMA and lambda protein phosphatase (sc-200312A) treated (**C**,**F**) K-562 nuclear extracts. Antibodies tested include p-B-Myb (H-3): sc-377500 (**A**,**B**,**C**) and B-Myb (C-20): sc-725 (**D**,**E**,**F**).



B-Myb (C-20): sc-725. Immunofluorescence staining of methanol-fixed HeLa cells showing nuclear and cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Zhang, H.S., et al. 2000. Exit from G_1 and S phase of cell cycle is regulated by repressor complexes containing DAC-Rb-hSWI/ SNF and Rb-hSWi/SNF. Cell 101: 79-89.
2. Heckman, C.A., et al. 2000. A-Myb up-regulates Bcl-2 through a Cdx binding site in t(14;18) lymphoma cells. J. Biol. Chem. 275: 6499-6508.
3. Cicchillitti, L., et al. 2004. B-Myb acts as a repressor of human COL1A1 collagen gene expression by interacting with Sp1 and CBF factors in scleroderma fibroblasts. Biochem. J. 378: 609-616.
4. Plante, I., et al. 2007. Sexual dimorphism in the regulation of liver connexin32 transcription in hexachlorobenzene-treated rats. Toxicol. Sci. 96: 47-57.
5. Corsino, P., et al. 2009. A novel class of cyclin-dependent kinase inhibitors identified by molecular docking act through a unique mechanism. J. Biol. Chem. 284: 29945-29955.
6. Huang, Y., et al. 2011. B-MYB delays cell aging by repressing $p16^{INK4a}$ transcription. Cell. Mol. Life Sci. 68: 893-901.
7. Zhan, M., et al. 2012. The B-MYB transcriptional network guides cell cycle progression and fate decisions to sustain self-renewal and the identity of pluripotent stem cells. PLoS ONE 7: e42350.

RESEARCH USE

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