SANTA CRUZ BIOTECHNOLOGY, INC.

Mxi1 (MXI1C2a): sc-130627



BACKGROUND

It is now well established that Myc regulation of cell proliferation and differentiation involves a family of related transcription factors. One such factor, Max, is an obligate heterodimeric partner for Myc and can also form heterodimers with at least four related proteins designated Mad 1, Mxi1 (also designated Mad 2), Mad 3 and Mad 4. Like Mad 1 and Mxi1, association of Mad 3 and Mad 4 with Max results in transcriptional repression. Both Myc and the Mad proteins have short half-lives and their synthesis is tightly regulated, while Max expression is constitutive and relatively stable. Two related mammalian cDNAs have been identified and shown to encode Madbinding proteins. Both possess sequence homology with the yeast transcription repressor Sin3 including four conserved paired amphipathic helix (PAH) domains. mSin3A and mSin3B specifically interact with the Mad proteins via their second paired amphipathic helix domain (PAH2). It has been suggested that Mad-Max heterodimers repress transcription by tethering mSin3 to DNA as corepressors.

REFERENCES

- Mukherjee, B., Morgerbesser S.D. and DePinho, R.A. 1992. Myc family oncoproteins function through a common pathway to transform normal cells in culture: cross-interference by Max and transacting dominant mutants. Genes Dev. 6: 1480-1492.
- Kretzner, L., Blackwood, E.M. and Eisenman, R.N. 1992. The Myc and Max proteins possess distinct transcriptional activities. Nature 359: 426-429.
- Ayer, D.E., Kretzner, L. and Eisenman, R.N. 1993. Mad: a heterodimeric partner for Max that antagonizes Myc transcriptional activity. Cell 72: 211-222.
- Amati, B., Littlewood, T.D., Evan, G.I. and Land, H. 1993. The c-Myc protein induces cell cycle progression and apoptosis through dimerization with Max. EMBO J. 12: 5083-5087.
- Ayer, D.E., Lawrence, Q.A. and Eisenman, R.N. 1995. Mad-Max transcriptional repression is mediated by ternary complex formation with mammalian homologs of yeast repressor Sin3. Cell 80: 767-776.

CHROMOSOMAL LOCATION

Genetic locus: MXI1 (human) mapping to 10q25.2.

SOURCE

Mxi1 (MXI1C2a) is a mouse monoclonal antibody raised against a recombinant protein corresponding to the C-terminus of Mxi1 of human origin.

PRODUCT

Each vial contains 100 $\mu g\, lgG_1$ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 1.0% stabilizer protein.

STORAGE

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

APPLICATIONS

Mxi1 (MXI1C2a) is recommended for detection of Mxi1 of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)].

Suitable for use as control antibody for Mxi1 siRNA (h): sc-35835, Mxi1 shRNA Plasmid (h): sc-35835-SH and Mxi1 shRNA (h) Lentiviral Particles: sc-35835-V.

Molecular Weight of Mxi1 isoforms: 26/22/33/21 kDa.

Positive Controls: U-937 cell lysate: sc-2239, IMR-32 cell lysate: sc-2409 or K-562 whole cell lysate: sc-2203.

RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz[®] Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml).

DATA



Mxi1 (MXI1C2a): sc-130627. Western blot analysis of human recombinant Mxi1 fusion protein.

SELECT PRODUCT CITATIONS

- Xu, L., Long, J., Wang, P., Liu, K., Mai, L. and Guo, Y. 2015. Association between the ornithine decarboxylase G316A polymorphism and breast cancer survival. Oncol. Lett. 10: 485-491.
- Dong, L., Liu, X., Wu, B., Li, C., Wei, X., Wumaier, G., Zhang, X., Wang, J., Xia, J., Zhang, Y., Yiminniyaze, R., Zhu, N., Li, J., Zhou, D., Zhang, Y., Li, S., Lv, J. and Li, S. 2022. Mxi1-0 promotes hypoxic pulmonary hypertension via ERK/c-Myc-dependent proliferation of arterial smooth muscle cells. Front. Genet. 13: 810157.

RESEARCH USE

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

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.