SANTA CRUZ BIOTECHNOLOGY, INC.

MLK3 (H-300): sc-13072

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

As a result of the binding of growth factors to their membrane receptors, cytoplasmic proteins containing Src homology 2 (SH2) domains associate with specific phosphotyrosine residues within the activated receptors and function as signaling intermediates. The action of such SH2 domain proteins frequently involves the activation of a second group of signaling intermediates characterized by SH3 domains. These latter proteins function through binding proline-rich sequences in target proteins. A novel human non-receptor protein kinase, designated either MLK3 or PTK1, is 847 amino acids in length and contains an SH3 domain in the absence of an SH2 domain. In addition, MLK3 is characterized by a leucine zipper basic region (a motif commonly associated with transcription factors) and has a long carboxy-terminal tail which exhibits proline-rich motifs similar to known SH3 binding sites. MLK3 is expressed widely and is related to the previously described MLK1 and MLK2 kinases.

CHROMOSOMAL LOCATION

Genetic locus: MAP3K11 (human) mapping to 11q13.1; Map3k11 (mouse) mapping to 19 A.

SOURCE

MLK3 (H-300) is a rabbit polyclonal antibody raised against amino acids 548-847 mapping at the C-terminus of MLK3 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.

APPLICATIONS

MLK3 (H-300) is recommended for detection of MLK3 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).

Suitable for use as control antibody for MLK3 siRNA (h): sc-35945, MLK3 siRNA (m): sc-35946, MLK3 shRNA Plasmid (h): sc-35945-SH, MLK3 shRNA Plasmid (m): sc-35946-SH, MLK3 shRNA (h) Lentiviral Particles: sc-35945-V and MLK3 shRNA (m) Lentiviral Particles: sc-35946-V.

Molecular Weight of MLK3: 95 kDa.

Positive Controls: MLK3 (h): 293 Lysate: sc-111047, MLK3 (m): 293T Lysate: sc-121686 or Jurkat whole cell lysate: sc-2204.

STORAGE

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

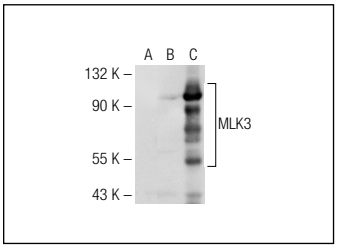
PROTOCOLS

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

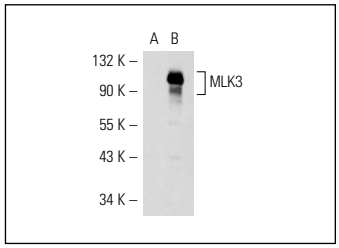
RESEARCH USE

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

DATA



MLK3 (H-300): sc-13072. Western blot analysis of MLK3 expression in non-transfected 293T: sc-117752 (**A**), mouse MLK3 transfected 293T: sc-121686 (**B**) and Jurkat (**C**) whole cell lysates.



MLK3 (H-300): sc-13072. Western blot analysis of MLK3 expression in non-transfected: sc-110760 (**A**) and human MLK3 transfected: sc-111047 (**B**) 293 whole cell lysates.

SELECT PRODUCT CITATIONS

1. Tian, H., et al. 2003. Antioxidant N-acetylcysteine and AMPA/KA receptor antagonist DNQX inhibited mixed lineage kinase-3 activation following cerebral ischemia in rat hippocampus. Neurosci. Res. 47: 47-53.
2. Chen, J., et al. 2009. GluR6-containing KA receptor mediates the activation of p38 MAP kinase in rat hippocampal CA1 region during brain ischemia injury. Hippocampus 19: 79-89.
3. Du, Y., et al. 2009. Neuroprotection of preconditioning against ischemic brain injury in rat hippocampus through inhibition of the assembly of GluR6-PSD95-mixed lineage kinase 3 signaling module via nuclear and non-nuclear pathways. Neuroscience 161: 370-380.
4. Li, T., et al. 2009. Inhibition of cerebral ischemia/reperfusion-induced injury by adenovirus expressed C-terminal amino acids of GluR6. Brain Res. 1300: 169-176.
5. Qi, S.H., et al. 2010. Neuroprotection of ethanol against ischemia/reperfusion-induced brain injury through decreasing c-Jun N-terminal kinase 3 (JNK3) activation by enhancing GABA release. Neuroscience 167: 1125-1137.
6. Li, C., et al. 2010. Coactivation of GABA receptors inhibits the JNK3 apoptotic pathway via disassembly of GluR6-PSD-95-MLK3 signaling module in KA-induced seizure. Epilepsia 51: 391-403.
7. Zhu, Q.J., et al. 2012. SUMOylation of the kainate receptor subunit GluK2 contributes to the activation of the MLK3-JNK3 pathway following kainate stimulation. FEBS Lett. 586: 1259-1264.