SANTA CRUZ BIOTECHNOLOGY, INC.

MEK-4 (K-18): sc-964

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

A family of protein kinases located upstream of the MAP kinases and responsible for their activation has been identified. The prototype member of this family, designated MAP kinase kinase or MEK-1, specifically phosphorylates the MAP kinase regulatory threonine and tyrosine residues present in the Thr-Glu-Tyr motif of ERK. A second MEK family member, MEK-2, resembles MEK-1 in its substrate specificity. MEK-3 (or MKK-3) functions to activate p38 MAP kinase, and MEK-4 (also called SEK1 or MKK-4) activates both p38 and JNK MAP kinases. MEK-5 appears to specifically phosphorylate ERK5, whereas MEK-6 phosphorylates p38 and p38b. MEK-7 (or MKK-7) phosphorylates and activates the JNK signal transduction pathway.

CHROMOSOMAL LOCATION

Genetic locus: MAP2K4 (human) mapping to 17p12; Map2k4 (mouse) mapping to 11 B3.

SOURCE

MEK-4 (K-18) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping near the N-terminus of MEK-4 of mouse origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-964 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

MEK-4 (K-18) is recommended for detection of MEK-4 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).

MEK-4 (K-18) is also recommended for detection of MEK-4 in additional species, including canine and bovine.

Suitable for use as control antibody for MEK-4 siRNA (h): sc-35909, MEK-4 siRNA (m): sc-35910, MEK-4 shRNA Plasmid (h): sc-35909-SH, MEK-4 shRNA Plasmid (m): sc-35910-SH, MEK-4 shRNA (h) Lentiviral Particles: sc-35909-V and MEK-4 shRNA (m) Lentiviral Particles: sc-35910-V.

Molecular Weight of MEK-4: 45 kDa.

Positive Controls: NIH/3T3 whole cell lysate: sc-2210 or Src-3T3 whole cell lysate.

RESEARCH USE

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

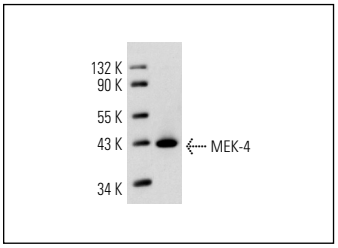
PROTOCOLS

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

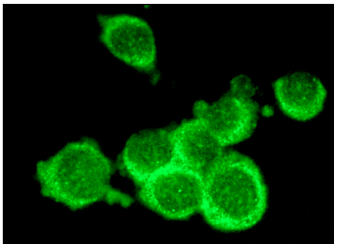
STORAGE

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

DATA



MEK-4 (K-18): sc-964. Western blot analysis of MEK-4 expression in Src-3T3 whole cell lysate.



MEK-4 (K-18): sc-964. Immunofluorescence staining of methanol-fixed K-562 cells showing cytoplasmic and membrane staining.

SELECT PRODUCT CITATIONS

1. Teramoto, H., et al. 1996. Signaling from the small GTP-binding proteins Rac 1 and Cdc42 to the c-Jun N-terminal kinase/stress-activated protein kinase pathway. A role for mixed lineage kinase 3/protein-tyrosine kinase 1, a novel member of the mixed lineage kinase family. J. Biol. Chem. 271: 27225-27228.
2. Xu, L., et al. 2008. Anthrax lethal toxin increases superoxide production in murine neutrophils via differential effects on MAPK signaling pathways. J Immunol. 180: 4139-4147.
3. Wen, X.R., et al. 2008. Dual inhibitory roles of geldanamycin on the c-Jun NH_2-terminal kinase 3 signal pathway through suppressing the expression of mixed-lineage kinase 3 and attenuating the activation of apoptosis signal-regulating kinase 1 via facilitating the activation of Akt in ischemic brain injury. Neuroscience 156: 483-497.
4. Lehmann, M., et al. 2009. Lung epithelial injury by *B. anthracis* lethal toxin is caused by MKK-dependent loss of cytoskeletal integrity. PLoS ONE 4: e4755.
5. deCathelineau, A.M. and Bokoch, G.M. 2009. Inactivation of rho GTPases by statins attenuates anthrax lethal toxin activity. Infect. Immun. 77: 348-359.
6. Harding, S.J., et al. 2010. Activation of ASK1, downstream MAPKK and MAPK isoforms during cardiac ischaemia. Biochim. Biophys. Acta 1802: 733-740.
7. Guo, Y., et al. 2013. Receptor for activated C kinase 1 promotes hepatocellular carcinoma growth by enhancing mitogen-activated protein kinase kinase 7 activity. Hepatology 57: 140-151.