SANTA CRUZ BIOTECHNOLOGY, INC.

MEK-3 (C-19): sc-961

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 Mkk3) functions to activate p38 MAP kinase, and MEK-4 (also called SEK1 or Mkk4) activates both p38 and JNK MAP kinases. MEK-5 appears to specifically phosphorylate ERK 5, whereas MEK-6 phosphorylates p38 and p38b. MEK-7 (or Mkk7) phosphorylates and activates the JNK signal transduction pathway.

CHROMOSOMAL LOCATION

Genetic locus: MAP2K3 (human) mapping to 17p11.2, MAP2K6 (human) mapping to 17q24.3; Map2k3 (mouse) mapping to 11 B2, Map2k6 (mouse) mapping to 11 E2.

SOURCE

MEK-3 (C-19) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of MEK-3 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.

Blocking peptide available for competition studies, sc-961 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

MEK-3 (C-19) is recommended for detection of MEK-3 and, to a lesser extent, MEK-6 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), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

MEK-3 (C-19) is also recommended for detection of MEK-3, and to a lesser extent, MEK-6 in additional species, including equine, canine and bovine.

Suitable for use as control antibody for MEK-3/6 siRNA (h): sc-43924, MEK-3/6 shRNA Plasmid (h): sc-43924-SH and MEK-3/6 shRNA (h) Lentiviral Particles: sc-43924-V.

Molecular Weight of MEK-3: 40 kDa.

Positive Controls: Jurkat whole cell lysate: sc-2204, K-562 whole cell lysate: sc-2203 or HeLa whole cell lysate: sc-2200.

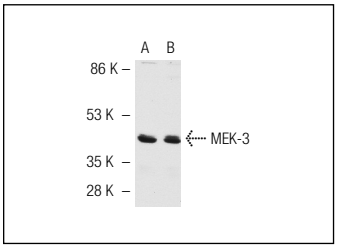
STORAGE

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

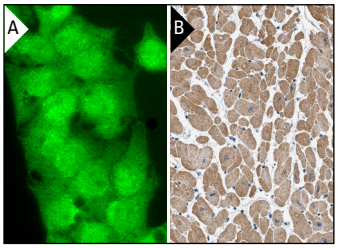
RESEARCH USE

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

DATA



MEK-3 (C-19): sc-961. Western blot analysis of MEK-3 expression in Jurkat (**A**) and K-562 (**B**) whole cell lysates.



MEK-3 (C-19): sc-961. Immunofluorescence staining of formalin-fixed HepG2 cells showing nuclear and cytoplasmic localization (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human heart muscle tissue showing cytoplasmic staining of myocytes. Kindly provided by The Swedish Human Protein Atlas (HPA) program (**B**).

SELECT PRODUCT CITATIONS

1. Ganiatsas, S., et al. 1998. SEK1 deficiency reveals mitogen-activated protein kinase cascade crossregulation and leads to abnormal hepatogenesis. Proc. Natl. Acad. Sci. USA 95: 6881-6886.
2. Panchal, R.G., et al. 2005. Purified *Bacillus anthracis* lethal toxin complex formed *in vitro* and during infection exhibits functional and biological activity. J. Biol. Chem. 280: 10834-10839.
3. Alvarez, M.E., et al. 2006. Neutrophil signaling pathways activated by bacterial DNA stimulation. J. Immunol. 177: 4037-4046.
4. Sun, P., et al. 2007. PRAK is essential for Ras-induced senescence and tumor suppression. Cell 128: 295-308.
5. Koçer, S.S., et al. 2008. Effects of anthrax lethal toxin on human primary keratinocytes. J. Appl. Microbiol. 105: 1756-1767.
6. Liu, S., et al. 2008. Matrix metalloproteinase-activated anthrax lethal toxin demonstrates high potency in targeting tumor vasculature. J. Biol. Chem. 283: 529-540.
7. 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.
8. Liu, S., et al. 2009. Capillary morphogenesis protein-2 is the major receptor mediating lethality of anthrax toxin *in vivo*. Proc. Natl. Acad. Sci. USA 106: 12424-12429.