SANTA CRUZ BIOTECHNOLOGY, INC.

p-MEK-4 (Thr 261): sc-7990

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 ERK-5, whereas MEK-6 phosphorylates p38 and p38β. MEK-7 (or MKK-7) phosphorylates and activates the JNK signal transduction pathway.

REFERENCES

1. Crews, C.M., et al. 1992. The primary structure of MEK, a protein kinase that phosphorylates the ERK gene product. Science 258: 478-480.
2. Wu, J., et al. 1993. Identification and characterization of a new mammalian mitogen-activated protein kinase kinase, MKK2. Mol. Cell Biol. 13: 4539-4548.
3. Zhou, G., et al. 1995. Components of a new human protein kinase signal transduction pathway. J. Biol. Chem. 270: 12665-12669.
4. Derijard, B., et al. 1995. Independent human MAP-kinase signal transduction pathways defined by MEK and MKK isoforms. Science 267: 682-685.
5. Jiang, Y., et al. 1996. Characterization of the structure and function of a new mitogen-activated protein kinase (p38β). J. Biol. Chem. 271: 17920-17926.

CHROMOSOMAL LOCATION

Genetic locus: MAP2K4 (human) mapping to 17p11.2; Map2k4 (mouse) mapping to 11 B3.

SOURCE

p-MEK-4 (Thr 261) is available as either goat (sc-7990) or rabbit (sc-7990-R) polyclonal affinity purified antibody raised against a short amino acid sequence containing phosphorylated Thr 261 of MEK-4 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-7990 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

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.

APPLICATIONS

p-MEK-4 (Thr 261) is recommended for detection of Thr 261 phosphorylated MEK-4 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).

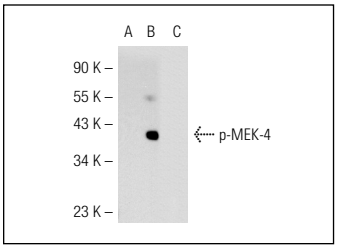
p-MEK-4 (Thr 261) is also recommended for detection of correspondingly phosphorylated Thr on MEK-4 in additional species, including equine, canine, bovine, porcine and avian.

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.

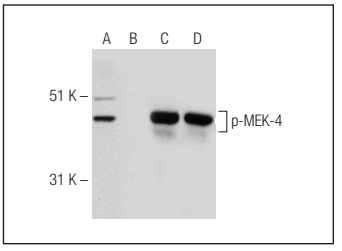
Molecular Weight of p-MEK-4: 45 kDa.

Positive Controls: UV irradiated HEK293 whole cell lysate or NIH/3T3 whole cell lysate: sc-2210.

DATA



p-MEK-4 (Thr 261)-R: sc-7990-R. Western blot analysis of MEK-4 phosphorylation in untreated (**A**), UV irradiated (**B**) and lambda protein phosphatase (sc-200312A) treated and UV irradiated (**C**) HEK293 whole cell lysates.



Western blot analysis of MEK-4 phosphorylation in untreated (**A**,**C**) and lambda protein phosphatase (sc-200312A) treated (**B**,**D**) rat brain tissue extracts. Antibodies tested include p-MEK-4 (Thr 261)-R: sc-7990-R (**A**,**B**) and MEK-4 (G-6): sc-166168 (**C**,**D**).

SELECT PRODUCT CITATIONS

1. Andreone, T.L., et al. 2003. Poly(ADP-ribose) polymerase-1 regulates activation of activator protein-1 in murine fibroblasts. J. Immunol. 170: 2113-2120.
2. Eminel, S., et al. 2004. JNK2 translocates to the mitochondria and mediates cytochrome c release in PC-12 cells in response to 6-hydroxydopamine. J. Biol. Chem. 279: 55385-55392.
3. Hata, N., et al. 2008. Increased expression of IRE1α and stress-related signal transduction proteins in ischemia-reperfusion injured retina. Clin. Ophthalmol. 2: 743-752.
4. Lagadinou, E.D., et al. 2008. c-Jun N-terminal kinase activation failure is a new mechanism of anthracycline resistance in acute myeloid leukemia. Leukemia 22: 1899-1908.

RESEARCH USE

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