SANTA CRUZ BIOTECHNOLOGY, INC.

p-ERK 1/2 (Thr 202): sc-101760

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The activation of signal transduction pathways by growth factors, hormones and neurotransmitters is mediated through two closely related MAP kinases, p44 and p42, designated extracellular-signal related kinase 1 (ERK 1) and ERK 2, respectively. ERK proteins are regulated by dual phosphorylation at Tyrosine 204 and 187 and Threonine 177 and 160 residues mapping within a characteristic Thr-Glu-Tyr motif. Phosphorylation at both the Threonine 202 and Tyrosine 204 residues of ERK 1 and Threonine 185 and Tyrosine 187 residues of ERK 2 is required for full enzymatic activation. The structural consequences of dual phosphorylation in ERK 2 include active site closure, alignment of key catalytic residues that interact with ATP, and remodeling of the activation loop. In response to activation, MAP kinases phosphorylate downstream components on serine and threonine. Upstream MAP kinase regulators include MAP kinase kinase (MEK), MEK kinase and Raf-1. The ERK family has three additional members: ERK 3, ERK 5 and ERK 6.

CHROMOSOMAL LOCATION

Genetic locus: MAPK3 (human) mapping to 16p11.2, MAPK1 (human) mapping to 22q11.2; Mapk3 (mouse) mapping to 7 F3, Mapk1 (mouse) mapping to 16 A3.

SOURCE

p-ERK 1/2 (Thr 202) is a rabbit polyclonal antibody raised against a short amino acid sequence containing Thr 202 phosphorylated ERK 1/2 of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

p-ERK 1/2 (Thr 202) is recommended for detection of Thr 202 phosphorylated ERK 1 of human origin and correspondingly phosphorylated Thr 203 of mouse and rat origin; and Thr 184 phosphorylated ERK 2 of human origin and correspondingly phosphorylated Thr 183 of mouse and rat 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 immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500).

Molecular Weight of p-ERK 1: 44 kDa.

Molecular Weight of p-ERK 2: 42 kDa.

Positive Controls: NIH/3T3 whole cell lysate: sc-2210, Jurkat whole cell lysate: sc-2204 or HeLa whole cell lysate: sc-2200.

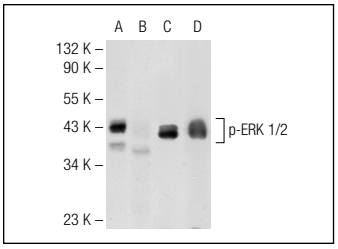
STORAGE

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

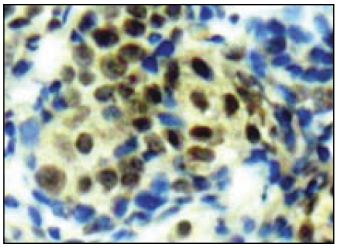
RESEARCH USE

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

DATA



Western blot analysis of ERK 1/2 phosphorylation in untreated (**A**,**C**), and lambda protein phosphatase (sc-200312A) treated (**B**,**D**) HeLa whole cell lysates. Antibodies tested include p-ERK 1/2 (Thr 202): sc-101760 (**A**,**B**) and ERK 2 (K-23): sc-153 (**C**,**D**).



p-ERK 1/2 (Thr 202): sc-101760. Immunoperoxidase staining of formalin-fixed, paraffin-embedded human breast carcinoma tissue showing nuclear and cytoplasmic staining.

SELECT PRODUCT CITATIONS

1. Lee, M.J., et al. 2008. Octylphenol stimulates resistin gene expression in 3T3-L1 adipocytes via the estrogen receptor and extracellular signal-regulated kinase pathways. Am. J. Physiol. Cell Physiol. 294: C1542-C1551.
2. Arora, D., et al. 2011. Protein-tyrosine phosphatase DEP-1 controls receptor tyrosine kinase FLT3 signaling. J. Biol. Chem. 286: 10918-10929.
3. De Vitis, S., et al. 2011. Tyr phosphatase-mediated P-ERK inhibition suppresses senescence in EIA + v-raf transformed cells, which, paradoxically, are apoptosis-protected in a MEK-dependent manner. Neoplasia 13: 120-130.
4. Lee, C.C., et al. 2011. Squamocin modulates histone H3 phosphorylation levels and induces G_1 phase arrest and apoptosis in cancer cells. BMC Cancer 11: 58.
5. Chen, X., et al. 2011. Houttuynia cordata blocks HSV infection through inhibition of NF-κB activation. Antiviral Res. 92: 341-345.
6. Impellizzeri, D., et al. 2011. Effect of apocynin, a NADPH oxidase inhibitor, on acute lung inflammation. Biochem. Pharmacol. 81: 636-648.
7. Jimenez-Vergara, A.C., et al. 2011. Influence of glycosaminoglycan identity on vocal fold fibroblast behavior. Acta Biomater 7: 3964-3972.
8. Giagulli, C., et al. 2011. Opposite effects of HIV-1 p17 variants on PTEN activation and cell growth in B cells. PLoS ONE 6: e17831.
9. Caccuri, F., et al. 2012. HIV-1 matrix protein p17 promotes angiogenesis via chemokine receptors CXCR1 and CXCR2. Proc. Natl. Acad. Sci. USA 109: 14580-14585.