SANTA CRUZ BIOTECHNOLOGY, INC.

JIP-1 (E-19): sc-7147

SANTA CRUZ BIOTECHNOLOGY
The Power to Question

BACKGROUND

c-Jun NH_2-terminal kinases (JNKs) are distant members of the MAP kinase family. JNK1 is activated by dual phosphorylation at a Thr-Pro-Tyr motif in response to ultraviolet (UV) light, and it functions to phosphorylate c-Jun at amino terminal serine regulatory sites, Ser 63 and Ser 73, resulting in transcriptional activation. Two additional JNK family members have been identified as JNK2 and JNK3. JIP-1 (for JNK interacting protein-1) has been 0identified as a cytoplasmic inhibitor of JNK that retains JNK in the cytoplasm, thereby inhibiting JNK-regulated gene expression. Evidence suggests that JNK1 and JNK2 bind to JIP-1 with greater affinity than to ATF-2 and c-Jun, which are targets of the JNK signaling pathway. JIP-1 contains an amino terminal JNK binding domain and a carboxy terminal SH3 domain. ATF-2 and c-Jun also contain the JNK binding domain and are thought to compete with JIP-1 for JNK binding. Multiple splice variants of JIP-1, including JIP-1b, JIP-1c (also designated islet-brain 1 or IB-1), JIP-2a, JIP-2b and JIP-3, have been identified in brain.

REFERENCES

1. Pulverer, B.J., et al. 1991. Phosphorylation of c-Jun mediated by MAP kinases. Nature 353: 670-674.
2. Smeal, T., et al. 1992. Oncoprotein-mediated signalling cascade stimulates c-Jun activity by phosphorylation of serines 63 and 73. Mol. Cell. Biol. 12: 3507-3512.
3. Derijard, B., et al. 1994. JNK1: a protein kinase stimulated by UV light and Ha-Ras that binds and phosphorylates the c-Jun activation domain. Cell 76: 1025-1037.
4. Kyriakis, J.M., et al. 1994. The stress-activated protein kinase subfamily of c-Jun kinases. Nature 369: 156-160.

CHROMOSOMAL LOCATION

Genetic locus: MAPK8IP1 (human) mapping to 11p11.2; Mapk8ip1 (mouse) mapping to 2 E1.

SOURCE

JIP-1 (E-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of JIP-1 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-7147 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.

RESEARCH USE

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

APPLICATIONS

JIP-1 (E-19) is recommended for detection of JIP-1 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), 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 JIP-1 siRNA (h): sc-35722, JIP-1 siRNA (m): sc-35723, JIP-1 shRNA Plasmid (h): sc-35722-SH, JIP-1 shRNA Plasmid (m): sc-35723-SH, JIP-1 shRNA (h) Lentiviral Particles: sc-35722-V and JIP-1 shRNA (m) Lentiviral Particles: sc-35723-V.

Molecular Weight of JIP-1: 115 kDa.

Positive Controls: rat cerebellum extract: sc-2398, mouse cerebellum extract: sc-2403 or PC-12 cell lysate: sc-2250.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunofluorescence: use donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

SELECT PRODUCT CITATIONS

1. Hashimoto, M., et al. 2002. α-synuclein protects against oxidative stress via inactivation of the c-Jun N-terminal kinase stress-signaling pathway in neuronal cells. J. Biol. Chem. 277: 11465.
2. Gdalyahu, A., et al. 2004. DCX, a new mediator of the JNK pathway. EMBO J. 23: 823-832.
3. Muresan, Z., et al. 2005. c-Jun NH_2-terminal kinase-interacting protein-3 facilitates phosphorylation and controls localization of Amyloid-β precursor protein. J. Neurosci. 25: 3741-3751.
4. Plaisance, V., et al. 2005. The repressor element silencing transcription factor (REST)-mediated transcriptional repression requires the inhibition of Sp1. J. Biol. Chem. 280: 401-407.

PROTOCOLS

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