SANTA CRUZ BIOTECHNOLOGY, INC.

c-Jun (79): sc-4113

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Genes belonging to the Jun and Fos oncogene families encode nuclear proteins that are found to be associated with a number of transcriptional complexes. The c-Jun protein is a major component of the transcription factor AP-1, originally shown to mediate phorbol ester tumor promoter (TPA)-induced expression of responsive genes through the TPA-response element (TRE). The Jun proteins form homo- and heterodimers which bind the TRE, but the Fos proteins are active only as heterodimers with any of the Jun proteins. Fos/Jun heterodimers have a much higher affinity for the TRE than Jun homodimers. Ha-Ras augments c-Jun activity and stimulates phosphorylation of its activation domain. An inhibitor of Fos/Jun function, termed IP-1, associates with Fos and Jun and is deactivated upon phosphorylation induced by the cAMP-dependent protein kinase A (PKA).

REFERENCES

1. Sambucetti, L.C. and Curran, T. 1986. The Fos protein complex is associated with DNA in isolated nuclei and binds to DNA cellulose. Science 234: 1417-1419.
2. Distel, R.J., et al. 1987. Nucleoprotein complexes that regulate gene expression in adipocyte differentiation: direct participation of c-Fos. Cell 49: 835-844.
3. Bohmann, D., et al. 1987. Human proto-oncogene c-Jun encodes a DNA binding protein with structural and functional properties of transcription factor AP-1. Science 238: 1386-1392.
4. Renz, M., et al. 1987. Chromatin association and DNA-binding properties of the c-Fos proto-oncogene product. Nucleic Acids Res. 15: 277-292.
5. Angel, P., et al. 1988. Oncogene Jun encodes a sequence-specific transactivator similar to AP-1. Nature 332: 166-171.
6. Franza, B.R., et al. 1988. The Fos complex and Fos related antigens recognize sequence elements that contain AP-1 binding sites. Science 239: 1150-1153.

CHROMOSOMAL LOCATION

Genetic locus: JUN (human) mapping to 1p32.1.

SOURCE

c-Jun (79) is produced in *E. coli* as a 37 kDa tagged fusion protein corresponding to amino acids 1-79 mapping within an amino-terminal domain of c-Jun of human origin.

PRODUCT

c-Jun (79) is purified (> 95%) by glutathione affinity chromatography; supplied as 50 µg protein in PBS containing 5 mM DTT and 50% glycerol.

APPLICATIONS

c-Jun (79) functions as a substrate for the JNK family of MAP kinases.

Also suitable as a Western blotting control for sc-822, sc-1694 and sc-7481.

Molecular Weight of c-Jun: 39 kDa.

STORAGE

Store at -20° C; stable for one year from the date of shipment.

SELECT PRODUCT CITATIONS

1. Coso, O.A., et al. 1995. The small GTP-binding proteins Rac1 and Cdc42 regulate the activity of the JNK/SAPK signaling pathway. Cell 81: 1137-1146.
2. Bendinelli, P., et al. 1996. The MAP kinase cascades are activated during post-ischemic liver reperfusion. FEBS Lett. 398: 193-197.
3. Vossler, M.R., et al. 1997. cAMP activates MAP kinase and Elk-1 through a B-Raf- and Rap1 dependent pathway. Cell 89: 73-82.
4. Lee, F.S., et al. 1998. MEKK1 activates both IκB kinase α and IκB kinase β. Proc. Natl. Acad. Sci. USA 95: 9506-9511.
5. Tanaka, M., et al. 1999. Embryonic lethality, liver degeneration, and impaired NFκB activation in IKK-β-deficient mice. Immunity 10: 421-429.
6. Maroni,P., et al. 2000. Cellular signalling after in vivo heat shock in the liver. Cell Biol. Int. 24: 145-152.
7. Watanabe, K., et al. 2001. Association of mitogen-activated protein kinase pathways with gingival epithelial cell responses to *Porphyromonas gingivalis* infection. Infect. Immun. 69: 6731-6737.
8. Minamino, T., et al. 2002. MEKK1 is essential for cardiac hypertrophy and dysfunction induced by G_q. Proc. Natl. Acad. Sci. USA 99: 3866-3871.
9. Jurewicz, A., et al. 2003. TNF-induced death of adult human oligodendrocytes is mediated by c-Jun NH_2-terminal kinase-3. Brain 126: 1358-1370.
10. Kim, S. 2004. Characterization of the prothrombin gene expression during nerve differentiation. Biochim. Biophys. Acta 1679: 1-9.
11. Vertrees, R.A., et al. 2005. A mechanism of hyperthermia-induced apoptosis in ras-transformed lung cells. Mol. Carcinog. 44: 111-121.
12. Jurewicz, A., et al. 2006. TRAIL-induced death of human adult oligodendrocytes is mediated by JNK pathway. Glia 53: 158-166.
13. Granja, A.G., et al. 2008. A238L inhibits NF-ATc2, NFκB, and c-Jun activation through a novel mechanism involving protein kinase C-θ-mediated up-regulation of the amino-terminal transactivation domain of p300. J. Immunol. 180: 2429-2442.
14. Beier, I., et al. 2008. Epidermal growth factor stimulates Rac1 and p21-activated kinase in vascular smooth muscle cells. Atherosclerosis 196: 92-97.
15. Granja, A.G., et al. 2009. African swine fever virus blocks the host cell antiviral inflammatory response through a direct inhibition of PKC-θ-mediated p300 transactivation. J. Virol. 83: 969-980.
16. Cetindere, T., et al. 2010. Induction of indoleamine 2, 3-dioxygenase by death receptor activation contributes to apoptosis of melanoma cells via mitochondrial damage-dependent ROS accumulation. Cell. Signal. 22: 197-211.

RESEARCH USE

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