SANTA CRUZ BIOTECHNOLOGY, INC.

JNK1 siRNA (m): sc-29381

BACKGROUND

c-Jun N-terminal kinases (JNKs) phosphorylate and augment transcriptional activity of c-Jun. JNKs originate from three genes that yield ten isoforms through alternative mRNA splicing, including JNK1α1,JNK1β1, JNK2α1, JNK2β1, and JNK3α1, which represent the p46 isoforms, and JNK1α2, JNK1β2, JNK2α2, JNK2β2, and JNK3β2, which represent the p54 isoforms. JNKs coordinate cell responses to stress and influence regulation of cell growth and transformation. The human JNK1 (PRKM8, SAPK1, MAPK8) gene maps to chromosome 10q11.22 and shares 83% amino acid identity with JNK2. JNK1 is necessary for normal activation and differentiation of CD4 helper T (TH) cells into TH1 and TH2 effector cells. Capsaicin activates JNK1 and p38 in Ras-transformed human breast epithelial cells. Nitrogen oxides (NOx) upregulate JNK1 in addition to c-Fos, c-Jun, and other signaling kinases, including MEKK1 and p38.

REFERENCES

1. Kallunki, T., et al. 1994. JNK2 contains a specificity-determining region responsible for efficient c-Jun binding and phosphorylation. Genes Dev. 8: 2996-3007.
2. Dong, C., et al. 1998. Defective T cell differentiation in the absence of JNK1. Science 282: 2092-2095.
3. Dong, C., et al. 2000. JNK is required for effector T-cell function but not for T-cell activation. Nature 405: 91-94.

CHROMOSOMAL LOCATION

Genetic locus: Mapk8 (mouse) mapping to 14 B.

PRODUCT

JNK1 siRNA (m) is a pool of 3 target-specific 19-25 nt siRNAs designed to knock down gene expression. Each vial contains 3.3 nmol of lyophilized siRNA, sufficient for a 10 µM solution once resuspended using protocol below. Suitable for 50-100 transfections. Also see JNK1 shRNA Plasmid (m): sc-29381-SH and JNK1 shRNA (m) Lentiviral Particles: sc-29381-V as alternate gene silencing products.

For independent verification of JNK1 (m) gene silencing results, we also provide the individual siRNA duplex components. Each is available as 3.3 nmol of lyophilized siRNA. These include: sc-29381A, sc-29381B and sc-29381C.

STORAGE AND RESUSPENSION

Store lyophilized siRNA duplex at -20° C with desiccant. Stable for at least one year from the date of shipment. Once resuspended, store at -20° C, avoid contact with RNAses and repeated freeze thaw cycles.

Resuspend lyophilized siRNA duplex in 330 µl of the RNAse-free water provided. Resuspension of the siRNA duplex in 330 µl of RNAse-free water makes a 10 µM solution in a 10 µM Tris-HCl, pH 8.0, 20 mM NaCl, 1 mM EDTA buffered solution.

APPLICATIONS

JNK1 siRNA (m) is recommended for the inhibition of JNK1 expression in mouse cells.

SUPPORT REAGENTS

For optimal siRNA transfection efficiency, Santa Cruz Biotechnology's siRNA Transfection Reagent: sc-29528 (0.3 ml), siRNA Transfection Medium: sc-36868 (20 ml) and siRNA Dilution Buffer: sc-29527 (1.5 ml) are recommended. Control siRNAs or Fluorescein Conjugated Control siRNAs are available as 10 µM in 66 µl. Each contain a scrambled sequence that will not lead to the specific degradation of any known cellular mRNA. Fluorescein Conjugated Control siRNAs include: sc-36869, sc-44239, sc-44240 and sc-44241. Control siRNAs include: sc-37007, sc-44230, sc-44231, sc-44232, sc-44233, sc-44234, sc-44235, sc-44236, sc-44237 and sc-44238.

GENE EXPRESSION MONITORING

JNK1 (F-3): sc-1648 is recommended as a control antibody for monitoring of JNK1 gene expression knockdown by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) or immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500).

RT-PCR REAGENTS

Semi-quantitative RT-PCR may be performed to monitor JNK1 gene expression knockdown using RT-PCR Primer: JNK1 (m)-PR: sc-29381-PR (20 µl, 497 bp). Annealing temperature for the primers should be 55-60° C and the extension temperature should be 68-72° C.

SELECT PRODUCT CITATIONS

1. Lu, C., et al. 2006. Cell apoptosis: requirement of H2AX in DNA ladder formation, but not for the activation of caspase-3. Mol. Cell 23: 121-132.
2. Guo, H., et al. 2008. Cyanidin 3-glucoside protects 3T3-L1 adipocytes against H_2O_2- or TNF-α-induced Insulin resistance by inhibiting c-Jun NH_2-terminal kinase activation. Biochem. Pharmacol. 75: 1393-1401.
3. Shin, D.M., et al. 2010. *Mycobacterium tuberculosis* eis regulates autophagy, inflammation, and cell death through redox-dependent signaling. PLoS Pathog. 6: e1001230.
4. Zhang, L., et al. 2011. TRAF2 phosphorylation promotes NFκB-dependent gene expression and inhibits oxidative stress-induced cell death. Mol. Biol. Cell 22: 128-140.
5. Wagley, Y., et al. 2013. Inhibition of c-Jun NH_2-terminal kinase stimulates μ opioid receptor expression via p38 MAPK-mediated nuclear NFκB activation in neuronal and non-neuronal cells. Biochim. Biophys. Acta 1833: 1476-1488.
6. Lee, K.G., et al. 2015. α-chaconine isolated from a *Solanum tuberosum L.* cv Jayoung suppresses lipopolysaccharide-induced pro-inflammatory mediators via AP-1 inactivation in RAW 264.7 macrophages and protects mice from endotoxin shock. Chem. Biol. Interact. 235: 85-94.
7. Kusuyama, J., et al. 2017. Constitutive activation of p46JNK2 is indispensable for C/EBPδ induction in the initial stage of adipogenic differentiation. Biochem. J. 474: 3421-3437.

RESEARCH USE

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