

I κ B- α (1-317): sc-4094

BACKGROUND

On the basis of both functional and structural considerations, members of the I κ B family of proteins can be divided into four groups. The first of these groups, I κ B- α , includes the avian protein pp40 and the mammalian MAD-3, both of which inhibit binding of p50-p65 NF κ B complex or Rel protein to their cognate binding sites but do not inhibit the binding of p50 homodimer to κ B sites, suggesting that the I κ B- α family binds to the p65 subunit of p50-p65 heterocomplex through ankyrin repeats. The second member of the I κ B family is represented by a protein designated I κ B- β . The third group of I κ B proteins is represented by I κ B- γ , which is identical in sequence with the C-terminal domain of the p110 precursor of NF κ B p50 and is expressed predominantly in lymphoid cells. An additional I κ B family member, I κ B- ϵ , has several phosphorylated forms and is primarily found complexed with Rel A and/or c-Rel.

REFERENCES

1. Ghosh, S., et al. 1990. Activation *in vitro* of NF κ B by phosphorylation of its inhibitor I κ B. *Nature* 344: 678-682.
2. Kerr, L.D., et al. 1991. The Rel-associated pp40 protein prevents DNA binding of Rel and NF κ B: relationship with I κ B- β and regulation by phosphorylation. *Genes Dev.* 5: 1464-1476.
3. Haskill, S., et al. 1991. Characterization of an immediate-early gene induced in adherent monocytes that encodes I B-like activity. *Cell* 65: 1281-1289.

CHROMOSOMAL LOCATION

Genetic locus: NFKBIA (human) mapping to 14q13.2; Nfkbia (mouse) mapping to 12 C1.

SOURCE

I κ B- α (1-317) is expressed in *E. coli* as a 62 kDa tagged fusion protein corresponding to full length (amino acids 1-317) I κ B- α of human origin.

PRODUCT

I κ B- α (1-317) is purified from bacterial lysates (> 98%) by glutathione agarose affinity chromatography; supplied as 50 μ g purified protein in PBS containing 0.1% azide, 5mM DTT and 50% glycerol.

Available as a Western blotting control; 10 μ g in 0.1 ml SDS-PAGE loading buffer, I κ B- α (1-317): sc-4094 WB.

APPLICATIONS

I κ B- α (1-317): sc-4094 is provided as purified protein for use in protein binding studies.

I κ B- α (1-317): sc-4094 WB is suitable as a Western blotting control for sc-203, sc-371, sc-847 and sc-1643.

Molecular Weight of I κ B- α : 35-41 kDa.

STORAGE

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

RESEARCH USE

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

SELECT PRODUCT CITATIONS

1. Spiecker, M., et al. 1998. Differential regulation of endothelial cell adhesion molecule expression by nitric oxide donors and antioxidants. *J. Leukoc. Biol.* 63: 732-739.
2. Chen, F., et al. 1999. Nitric oxide inhibits HIV Tat-induced NF κ B activation. *Am. J. Pathology* 155: 275-284.
3. Castrillo, A., et al. 2000. Inhibition of I κ B kinase and I κ B phosphorylation by 15-deoxy- $\Delta^{12,14}$ -prostaglandin J₂ in activated murine macrophages. *Mol. Cell. Biol.* 20: 1692-1698.
4. Kurokouchi, K., et al. 2001. Oscillating fluid flow inhibits TNF- α -induced NF κ B activation via an I κ B kinase pathway in osteoblast-like UMR106 cells. *J. Biol. Chem.* 276: 13499-13504.
5. Kim, B.Y., et al. 2002. Constitutive activation of NF κ B in Ki-Ras-transformed prostate epithelial cells. *Oncogene* 21: 4490-4497.
6. Philip, S., et al. 2003. Osteopontin induces nuclear factor κ B-mediated promatrix metalloproteinase-2 activation through I κ B- α /IKK signaling pathways, and curcumin (diferulolylmethane) down-regulates these pathways. *J. Biol. Chem.* 278: 14487-14497.
7. Asehounne, K., et al. 2004. Involvement of reactive oxygen species in Toll-like receptor 4-dependent activation of NF κ B. *J. Immunol.* 172: 2522-2529.
8. Ruiz, P.A. and Haller, D. 2006. Functional diversity of flavonoids in the inhibition of the proinflammatory NF κ B, IRF, and Akt signaling pathways in murine intestinal epithelial cells. *J. Nutr.* 136: 664-671.
9. Vacca, A., et al. 2006. Notch3 and pre-TCR interaction unveils distinct NF κ B pathways in T-cell development and leukemia. *EMBO J.* 25: 1000-1008.
10. Planavila, A., et al. 2006. Inhibition of cardiac hypertrophy by triflusal (4-trifluoromethyl derivative of salicylate) and its active metabolite. *Mol. Pharmacol.* 69: 1174-1181.
11. Tang, C.H., et al. 2007. Basic fibroblast growth factor stimulates fibronectin expression through phospholipase C γ , protein kinase C α , c-Src, NF κ B, and p300 pathway in osteoblasts. *J. Cell. Physiol.* 211: 45-55.
12. Todd, M. K., et al. 2007. Thiazolidinediones enhance skeletal muscle triacylglycerol synthesis while protecting against fatty acid-induced inflammation and insulin resistance. *Am. J. Physiol. Endocrinol. Metab.* 29: E485-E493.
12. Fernández-Velasco, M., et al. 2012. NOD1 activation induces cardiac dysfunction and modulates cardiac fibrosis and cardiomyocyte apoptosis. *PLoS ONE* 7: e45260.

PROTOCOLS

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