SANTA CRUZ BIOTECHNOLOGY, INC.

IKAP (33): sc-136412

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The transcription factor NFκB is retained in the cytoplasm in an inactive form by the inhibitory protein IκB. Activation of NFκB requires that IκB be phosphorylated on specific Serine residues, which results in the targeted degradation of IκB. IκB kinase α (IKKα), previously designated CHUK, interacts with IκB-α and specifically phosphorylates IκB-α on the sites that trigger its degradation, serines 32 and 36. IKKα appears to be critical for NFκB activation in response to proinflammatory cytokines. Phosphorylation of the IκB by IKKα is stimulated by the NFκB inducing kinase (NIK), which itself is a central regulator for NFκB activation in response to TNF and IL-1. The functional IKK complex contains three subunits, IKKα, IKKβ and IKKγ (also designated NEMO) and each appears to make essential contributions to IκB phosphorylation. IKAP (IKK complex-associated protein) is a protein that acts as a scaffold, interacting with NIK, IKKα and IKKβ and assembling them into an active kinase complex.

REFERENCES

1. Verma, I.M., et al. 1995. Rel/NFκB/IκB family: intimate tales of association and dissociation. Genes Dev. 9: 2723-2735.
2. Thanos, D. and Maniatis, T. 1995. NFκB: a lesson in family values. Cell 80: 529-532.
3. Conelly, M.A. and Marcu, K.B.1995. CHUK, a new member of the helix-loop-helix and leucine zipper families of interacting proteins, contains a serine/threonine kinase catalytic domain. Cell. Mol. Biol. Res. 41: 537-549.
4. Malinin, N.L., et al. 1997. MAP3K-related kinase involved in NFκB induction by TNF, CD95 and IL-1. Nature 385: 540-544.
5. DiDonato, J.A., et al. 1997. A cytokine-responsive IκB kinase that activates the transcription factor NFκB. Nature 388: 548-554.
6. Régnier, C.H., et al. 1997. Identification and characterization of an IκB kinase. Cell 90: 373-383.

CHROMOSOMAL LOCATION

Genetic locus: IKBKAP (human) mapping to 9q31.3.

SOURCE

IKAP (33) is a mouse monoclonal antibody raised against amino acids 796-1008 of IKAP of human origin.

PRODUCT

Each vial contains 200 µg IgG_{2a} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

IKAP (33) is available conjugated to agarose (sc-136412 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; and to HRP (sc-136412 HRP), 200 µg/ml, for WB, IHC(P) and ELISA.

STORAGE

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

APPLICATIONS

IKAP (33) is recommended for detection of IKAP of human 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)] and immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500).

Suitable for use as control antibody for IKAP siRNA (h): sc-40692, IKAP shRNA Plasmid (h): sc-40692-SH and IKAP shRNA (h) Lentiviral Particles: sc-40692-V.

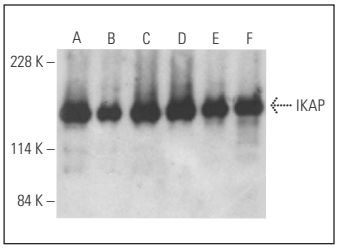
Molecular Weight of IKAP: 150 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200, HL-60 whole cell lysate: sc-2209 or Hep G2 cell lysate: sc-2227.

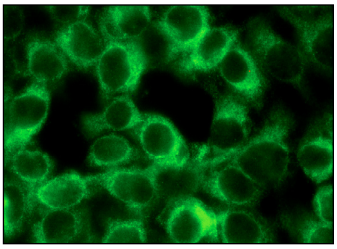
RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850.

DATA



IKAP (33) HRP: sc-136412 HRP. Direct western blot analysis of IKAP expression in Jurkat (**A**), HeLa (**B**), Hep G2 (**C**), K-562 (**D**), MCF7 (**E**) and HL-60 (**F**) whole cell lysates.



IKAP (33): sc-136412. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Bruun, G.H., et al. 2018. Blocking of an intronic splicing silencer completely rescues IKBKAP exon 20 splicing in familial dysautonomia patient cells. Nucleic Acids Res. 46: 7938-7952.
2. Morini, E., et al. 2019. ELP1 splicing correction reverses proprioceptive sensory loss in familial dysautonomia. Am. J. Hum. Genet. 12 pii: S0002-9297(19)30054-0.

RESEARCH USE

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