SANTA CRUZ BIOTECHNOLOGY, INC.

p-IKKγ (36.Ser 376): sc-293135

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The Power to Question

BACKGROUND

Activation of NFκB requires that IκB be phosphorylated on specific serine residues, which results in 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 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 appear to make essential contributions to IκB phosphorylation. The IKKβ phosphorylates human IKKγ at Ser 31, Ser 43, and Ser 376 following the enforced expression of either the Tax oncoprotein or the type 1 TNF receptor.

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., et al. 1995. NFκB: a lesson in family values. Cell 80: 529-532.
3. Conelly, M.A., et al. 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 Iκ-B kinase that activates the transcription factor NFκB. Nature 388: 548-554.
6. Regnier, C.H., et al. 1997. Identification and characterization of an IκB kinase. Cell 90: 373-383.
7. Zandi, E., et al. 1997. The IκB kinase complex (IKK) contains two kinase subunits, IKKα and IKKβ, necessary for IκB phosphorylation and NFκB activation. Cell 91: 243-252.
8. Song, H.Y., et al. 1997. Tumor necrosis factor (TNF)-mediated kinase cascades: bifurcation of nuclear factor-κB and c-Jun N-terminal kinase (JNK/SAPK) pathways at TNF receptor-associated factor 2. Proc. Natl. Acad. Sci. USA 94: 9792-9296.
9. Yamaoka, S., et al. 1998. Complementation cloning of NEMO, a component of the IκB kinase complex essential for NFκB activation. Cell 93: 1231-1240.

CHROMOSOMAL LOCATION

Genetic locus: IKBKG (human) mapping to Xq28.

SOURCE

p-IKKγ (36.Ser 376) is a mouse monoclonal antibody raised against a short amino acid sequence containing Ser 376 phosphorylated IKKγ of human origin.

RESEARCH USE

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

PRODUCT

Each vial contains 200 µg IgG_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

p-IKKγ (36.Ser 376) is available conjugated to agarose (sc-293135 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; and to HRP (sc-293135 HRP), 200 µg/ml, for WB, IHC(P) and ELISA.

APPLICATIONS

p-IKKγ (36.Ser 376) is recommended for detection of Ser 376 phosphorylated IKKγ of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for IKKα siRNA (h): sc-29365, IKKα shRNA Plasmid (h): sc-29365-SH and IKKα shRNA (h) Lentiviral Particles: sc-29365-V.

Molecular Weight of IKKγ: 48 kDa.

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, TBS Blotto B Blocking Reagent: sc-2335 (use 50 mM NaF, sc-24988, as diluent), Lambda Phosphatase: sc-200312A and Western Blotting Luminol Reagent: sc-2048.

SELECT PRODUCT CITATIONS

1. Lin, H.W., et al. 2014. Regulation of virus-induced inflammatory response by *Dunaliella salina* alga extract in macrophages. Food Chem. Toxicol. 71: 159-165.
2. Che, D.N., et al. 2020. Luteolin suppresses IL-31 production in IL-33-stimulated mast cells through MAPK and NFκB signaling pathways. Int. Immunopharmacol. 83: 106403.
3. Wu, C.C., et al. 2020. β-funaltrexamine displayed anti-inflammatory and neuroprotective effects in cells and rat model of stroke. Int. J. Mol. Sci. 21: 3866.
4. Wang, H., et al. 2021. Aspartate metabolism facilitates IL-1β production in inflammatory macrophages. Front. Immunol. 12: 753092.
5. Yilmaz, D.E., et al. 2023. NLRX1 ligand, docosahexaenoic acid, ameliorates LPS-induced inflammatory hyperalgesia by decreasing TRAF6/IKK/IκB-α/NF-κB signaling pathway activity. Cell. Mol. Biol. (Noisy-le-grand). 69: 15-23.

STORAGE

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