IKKγ (72C627): sc-56919



The Power to Question

BACKGROUND

The transcription factor NF κ B is retained in the cytoplasm in an inactive form by the inhibitory protein I κ B. Activation of NF κ B requires that I κ B be phospho-rylated on specific serine residues, which results in targeted degradation of I κ B. I κ B kinase α (IKK α), previously designated CHUK, interacts with I κ B α and specifically phosphorylates I κ B α on Serine 32 and 36, the sites that trigger its degradation. IKK α appears to be critical for NF κ B activation in response to proinflammatory cytokines. Phosphorylation of I κ B by IKK α is stimulated by the NF κ B inducing kinase (NIK), which itself is a central regulator for NF κ B activation in response to TNF and IL-1. The functional IKK complex contains three subunits, IKK α , IKK β and IKK γ (also designated NEMO), and each appear to make essential contributions to I κ B phosphorylation.

REFERENCES

- 1. Verma, I.M., et al. 1995. Rel/NF κ B/I κ B family: intimate tales of association and dissociation. Genes Dev. 9: 2723-2735.
- 2. Thanos, D., et al. 1995. NFkB: 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 NF κ B induction by TNF, CD95 and IL-1. Nature 385: 540-544.
- 5. DiDonato, J.A., et al. 1997. A cytokine-responsive $I\kappa B$ kinase that activates the transcription factor NF κB . Nature 388: 548-554.
- 6. Regnier, C.H., et al. 1997. Identification and characterization of an $l\kappa B$ kinase. Cell 90: 373-383.
- 7. Zandi, E., et al. 1997. The $I\kappa B$ kinase complex (IKK) contains two kinase subunits, $IKK\alpha$ and $IKK\beta$, necessary for $I\kappa B$ phosphorylation and $NF\kappa B$ activation. Cell 91: 243-252.

CHROMOSOMAL LOCATION

Genetic locus: IKBKG (human) mapping to Xq28.

SOURCE

IKK γ (72C627) is a mouse monoclonal antibody raised against full length His-tagged IKK γ of human origin.

PRODUCT

Each vial contains 100 μg lgG_1 in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

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

RESEARCH USE

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

APPLICATIONS

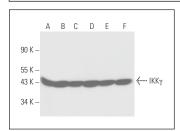
IKK γ (72C627) is recommended for detection of IKK γ of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

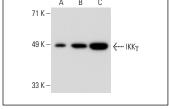
Suitable for use as control antibody for IKK γ siRNA (h): sc-29363, IKK γ shRNA Plasmid (h): sc-29363-SH and IKK γ shRNA (h) Lentiviral Particles: sc-29363-V.

Molecular Weight of IKKγ: 48 kDa.

Positive Controls: IKK γ (h): 293T Lysate: sc-116282, K-562 whole cell lysate: sc-2203 or HeLa whole cell lysate: sc-2200.

DATA





IKKy (72C627): sc-56919. Western blot analysis of IKKy expression in BJAB (A), Jurkat (B), K-562 (C), U-937 (D), SW-13 (E) and HeLa (F) whole cell lysates.

IKKy (72C627): sc-56919. Western blot analysis of IKKy expression in non-transfected 293T: sc-117752 (**A**), human IKKy transfected 293T: sc-116282 (**B**) and K-562 (**C**) whole

SELECT PRODUCT CITATIONS

- 1. Randall, C.M., et al. 2012. The MC159 protein from the molluscum contagiosum poxvirus inhibits NF κ B activation by interacting with the I κ B kinase complex. J. Immunol. 188: 2371-2379.
- Blakely, C.M., et al. 2015. NFκB-activating complex engaged in response to EGFR oncogene inhibition drives tumor cell survival and residual disease in lung cancer. Cell Rep. 11: 98-110.
- Zhou, Q., et al. 2016. Loss-of-function mutations in TNFAIP3 leading to A20 haploinsufficiency cause an early-onset autoinflammatory disease. Nat. Genet. 48: 67-73.
- Franco-Jarava, C., et al. 2018. TNFAIP3 haploinsufficiency is the cause of autoinflammatory manifestations in a patient with a deletion of 13Mb on chromosome 6. Clin. Immunol. 191: 44-51.
- 5. Mulhern, C.M., et al. 2019. Janus kinase 1/2 inhibition for the treatment of autoinflammation associated with heterozygous TNFAIP3 mutation. J. Allergy Clin. Immunol. 144: 863-866.e5.



See **IKK**Y **(F-10):** sc-166398 for IKKY antibody conjugates, including AC, HRP, FITC, PE, and Alexa Fluor® 488, 546, 594, 647, 680 and 790.