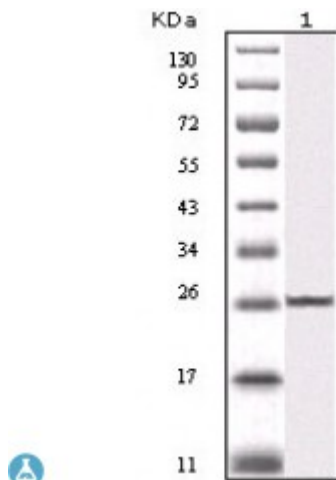


Anti-IK beta antibody



Description	Mouse monoclonal to IKKbeta.
Model	STJ98165
Host	Mouse
Reactivity	Human
Applications	ELISA, IHC, WB
Immunogen	Purified recombinant fragment of IKKbeta expressed in E. Coli.
Gene ID	3551
Gene Symbol	IKBKB
Dilution range	WB 1:500-1:2000IHC 1:200-1:1000ELISA 1:10000
Specificity	IKKbeta Monoclonal Antibody detects endogenous levels of IKKbeta protein.
Tissue Specificity	Highly expressed in heart, placenta, skeletal muscle, kidney, pancreas, spleen, thymus, prostate, testis and peripheral blood.
Purification	Affinity purification
Clone ID	10A2C5B3
Note	For Research Use Only (RUO).
Protein Name	Inhibitor of nuclear factor kappa-B kinase subunit beta I-kappa-B-kinase beta IKK-B IKK-beta IkbKB I-kappa-B kinase 2 IKK2 Nuclear factor NF-kappa-B inhibitor kinase beta NFKBIKB
Clonality	Monoclonal
Conjugation	Unconjugated

Isotype	IgG1
Formulation	Ascitic fluid containing 0.03% sodium azide.
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:5960OMIM:603258
Alternative Names	Inhibitor of nuclear factor kappa-B kinase subunit beta I-kappa-B-kinase beta IKK-B IKK-beta IkbKB I-kappa-B kinase 2 IKK2 Nuclear factor NF-kappa-B inhibitor kinase beta NFKBIKB
Function	Serine kinase that plays an essential role in the NF-kappa-B signaling pathway which is activated by multiple stimuli such as inflammatory cytokines, bacterial or viral products, DNA damages or other cellular stresses. Acts as part of the canonical IKK complex in the conventional pathway of NF-kappa-B activation and phosphorylates inhibitors of NF-kappa-B on 2 critical serine residues. These modifications allow polyubiquitination of the inhibitors and subsequent degradation by the proteasome. In turn, free NF-kappa-B is translocated into the nucleus and activates the transcription of hundreds of genes involved in immune response, growth control, or protection against apoptosis. In addition to the NF-kappa-B inhibitors, phosphorylates several other components of the signaling pathway including NEMO/IKBKG, NF-kappa-B subunits RELA and NFkB1, as well as IKK-related kinases TBK1 and IKBKE. IKK-related kinase phosphorylations may prevent the overproduction of inflammatory mediators since they exert a negative regulation on canonical IKKs. Phosphorylates FOXO3, mediating the TNF-dependent inactivation of this pro-apoptotic transcription factor. Also phosphorylates other substrates including NCOA3, BCL10 and IRS1. Within the nucleus, acts as an adapter protein for NFkBIA degradation in UV-induced NF-kappa-B activation.
Sequence and Domain Family	The kinase domain is located in the N-terminal region. The leucine zipper is important to allow homo- and hetero-dimerization. At the C-terminal region is located the region responsible for the interaction with NEMO/IKBKG.
Cellular Localization	Cytoplasm Nucleus Membrane raft. Colocalized with DPP4 in membrane rafts.
Post-translational Modifications	Upon cytokine stimulation, phosphorylated on Ser-177 and Ser-181 by MEKK1 and/or MAP3K14/NIK as well as TBK1 and PRKCZ; which enhances activity. Once activated, autophosphorylates on the C-terminal serine cluster; which decreases activity and prevents prolonged activation of the inflammatory response. Phosphorylated by the IKK-related kinases TBK1 and IKBKE, which is associated with reduced CHUK/IKKA and IKBKB activity and NF-kappa-B-dependent gene transcription. Dephosphorylated at Ser-177 and Ser-181 by PPM1A and PPM1B. (Microbial infection) Acetylation of Thr-180 by Yersinia yopJ prevents phosphorylation and activation, thus blocking the I-kappa-B pathway. Ubiquitinated. Monoubiquitination involves TRIM21 that leads to inhibition of Tax-induced NF-kappa-B signaling. According to PubMed:19675099, 'Ser-163' does not serve as a monoubiquitination site. According to PubMed:16267042, ubiquitination on 'Ser-163' modulates phosphorylation on C-terminal serine residues. (Microbial infection) Monoubiquitination by TRIM21 is disrupted by Yersinia yopJ. Hydroxylated by PHD1/EGLN2, loss of hydroxylation under hypoxic conditions results in activation of NF-kappa-B.

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