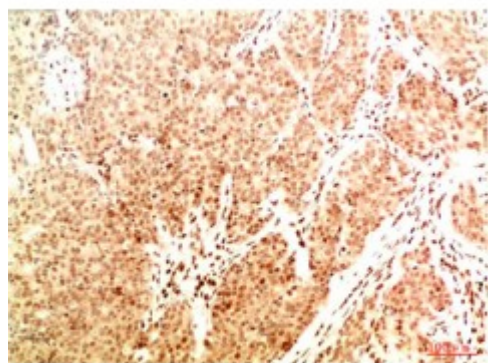


Anti-NF-kB P65 (Acetyl K314/K315) antibody



Description	Mouse monoclonal to NF-kB P65 (Acetyl K314/K315).
Model	STJ98905
Host	Mouse
Reactivity	Human, Mouse, Rat
Applications	ELISA, WB
Immunogen	synthetic peptide derived from Acetyl NF kB P65 (K314/K315).
Immunogen Region	Acetyl NF kB P65 K314/K315.
Gene ID	5970
Gene Symbol	RELA
Dilution range	WB 1:500-2000ELISA 1:10000-20000
Specificity	The antibody detects endogenous Acetyl NF kB P65 (K314/K315) protein.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Note	For Research Use Only (RUO).
Protein Name	Transcription factor p65 Nuclear factor NF-kappa-B p65 subunit Nuclear factor of kappa light polypeptide gene enhancer in B-cells 3
Molecular Weight	65kDa
Clonality	Monoclonal
Conjugation	Unconjugated

Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:9955OMIM:164014
Alternative Names	Transcription factor p65 Nuclear factor NF-kappa-B p65 subunit Nuclear factor of kappa light polypeptide gene enhancer in B-cells 3
Function	NF-kappa-B is a pleiotropic transcription factor present in almost all cell types and is the endpoint of a series of signal transduction events that are initiated by a vast array of stimuli related to many biological processes such as inflammation, immunity, differentiation, cell growth, tumorigenesis and apoptosis. NF-kappa-B is a homo- or heterodimeric complex formed by the Rel-like domain-containing proteins RELA/p65, RELB, NFKB1/p105, NFKB1/p50, REL and NFKB2/p52 and the heterodimeric p65-p50 complex appears to be most abundant one. The dimers bind at kappa-B sites in the DNA of their target genes and the individual dimers have distinct preferences for different kappa-B sites that they can bind with distinguishable affinity and specificity. Different dimer combinations act as transcriptional activators or repressors, respectively. NF-kappa-B is controlled by various mechanisms of post-translational modification and subcellular compartmentalization as well as by interactions with other cofactors or corepressors. NF-kappa-B complexes are held in the cytoplasm in an inactive state complexed with members of the NF-kappa-B inhibitor (I-kappa-B) family. In a conventional activation pathway, I-kappa-B is phosphorylated by I-kappa-B kinases (IKKs) in response to different activators, subsequently degraded thus liberating the active NF-kappa-B complex which translocates to the nucleus. NF-kappa-B heterodimeric p65-p50 and p65-c-Rel complexes are transcriptional activators. The NF-kappa-B p65-p65 complex appears to be involved in invasion-mediated activation of IL-8 expression. The inhibitory effect of I-kappa-B upon NF-kappa-B the cytoplasm is exerted primarily through the interaction with p65. p65 shows a weak DNA-binding site which could contribute directly to DNA binding in the NF-kappa-B complex. Associates with chromatin at the NF-kappa-B promoter region via association with DDX1. Essential for cytokine gene expression in T-cells .
Sequence and Domain Family	the 9aaTAD motif is a transactivation domain present in a large number of yeast and animal transcription factors.
Cellular Localization	Nucleus. Cytoplasm. Colocalized with DDX1 in the nucleus upon TNF-alpha induction . Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I-kappa-B). Colocalizes with GFI1 in the nucleus after LPS stimulation.
Post-translational Modifications	Ubiquitinated, leading to its proteasomal degradation. Degradation is required for termination of NF-kappa-B response. Monomethylated at Lys-310 by SETD6. Monomethylation at Lys-310 is recognized by the ANK repeats of EHMT1 and promotes the formation of repressed chromatin at target genes, leading to down-regulation of NF-kappa-B transcription factor activity. Phosphorylation at Ser-311 disrupts the interaction with EHMT1 without preventing monomethylation at Lys-310 and relieves the repression of target genes . Phosphorylation at Ser-311 disrupts the interaction with EHMT1 and promotes transcription factor activity . Phosphorylation on Ser-536 stimulates

acetylation on Lys-310 and interaction with CBP; the phosphorylated and acetylated forms show enhanced transcriptional activity. Phosphorylation at Ser-276 by RPS6KA4 and RPS6KA5 promotes its transactivation and transcriptional activities. Reversibly acetylated; the acetylation seems to be mediated by CBP, the deacetylation by HDAC3 and SIRT2. Acetylation at Lys-122 enhances DNA binding and impairs association with NFKBIA. Acetylation at Lys-310 is required for full transcriptional activity in the absence of effects on DNA binding and NFKBIA association. Acetylation at Lys-310 promotes interaction with BRD4. Acetylation can also lower DNA-binding and results in nuclear export. Interaction with BRMS1 promotes deacetylation of Lys-310. Lys-310 is deacetylated by SIRT2. S-nitrosylation of Cys-38 inactivates the enzyme activity. Sulfhydration at Cys-38 mediates the anti-apoptotic activity by promoting the interaction with RPS3 and activating the transcription factor activity. Sumoylation by PIAS3 negatively regulates DNA-bound activated NF-kappa-B. Proteolytically cleaved within a conserved N-terminus region required for base-specific contact with DNA in a CPEN1-mediated manner, and hence inhibits NF-kappa-B transcriptional activity .