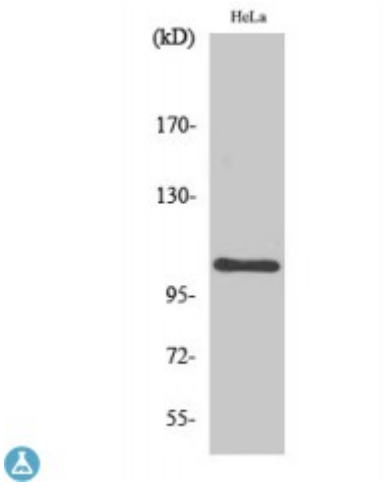
Anti-N kappa-p105 antibody



Description	Rabbit polyclonal to NFkappaB-p105.

Model	STJ94463
Host	Rabbit
Reactivity	Human, Mouse
Applications	ELISA, IF, IHC, WB
Immunogen	Synthesized peptide derived from human NFkappaB-p105 around the non-phosphorylation site of S927.
Immunogen Region	870-950 aa
Gene ID	4790
Gene Symbol	NFKB1
Dilution range	WB 1:500-1:2000IHC 1:100-1:300IF 1:200-1:1000ELISA 1:40000
Specificity	NFkappaB-p105 Polyclonal Antibody detects endogenous levels of NFkappaB-p105 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	Nuclear factor NF-kappa-B p105 subunit DNA-binding factor KBF1 EBP-1 Nuclear factor of kappa light polypeptide gene enhancer in B-cells 1 Nuclear factor NF-kappa-B p50 subunit
Molecular Weight	50/110 kDa

Clonality	Polyclonal
Conjugation	Unconjugated
Isotype	IgG
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:7794OMIM:164011
Alternative Names	Nuclear factor NF-kappa-B p105 subunit DNA-binding factor KBF1 EBP-1 Nuclear factor of kappa light polypeptide gene enhancer in B-cells 1 Nuclear factor NF-kappa-B p50 subunit
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 RelB-p50 complexes are transcriptional activators. The NF-kappa-B p50-p50 homodimer is a transcriptional repressor, but can act as a transcriptional activator when associated with BCL3. NFKB1 appears to have dual functions such as cytoplasmic retention of attached NF-kappa-B proteins by p105 and generation of p50 by a cotranslational processing. The proteasome-mediated process ensures the production of both p50 and p105 and preserves their independent function, although processing of NFKB1/p105 also appears to occur post-translationally. p50 binds to the kappa-B consensus sequence 5'-GGRNNYYCC-3', located in the enhancer region of genes involved in immune response and acute phase reactions. In a complex with MAP3K8, NFKB1/p105 represses MAP3K8-induced MAPK signaling; active MAP3K8 is released by proteasome-dependent degradation of NFKB1/p105.
Sequence and Domain Family	The C-terminus of p105 might be involved in cytoplasmic retention, inhibition of DNA-binding, and transcription activation.; Glycine-rich region (GRR) appears to be a critical element in the generation of p50.
Cellular Localization	Nucleus. Cytoplasm. Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I-kappa-B).

Post-translational Modifications	While translation occurs, the particular unfolded structure after the GRR repeat promotes the generation of p50 making it an acceptable substrate for the proteasome. This process is known as cotranslational processing. The processed form is active and the unprocessed form acts as an inhibitor (I kappa B-like), being able to form cytosolic complexes with NF-kappa B, trapping it in the cytoplasm. Complete folding of the region downstream of the GRR repeat precludes processing. Phosphorylation at 'Ser-903' and 'Ser-907' primes p105 for proteolytic processing in response to TNF-alpha stimulation. Phosphorylation at 'Ser-927' and 'Ser-932' are required for BTRC/BTRCP-mediated proteolysis. Polyubiquitination seems to allow p105 processing. S-nitrosylation of Cys-61 affects DNA binding. The covalent modification of cysteine by 15-deoxy-Delta12,14-prostaglandin-J2 is autocatalytic and reversible. It may occur as an alternative to other cysteine modifications, such as S-nitrosylation and S-palmitoylation.