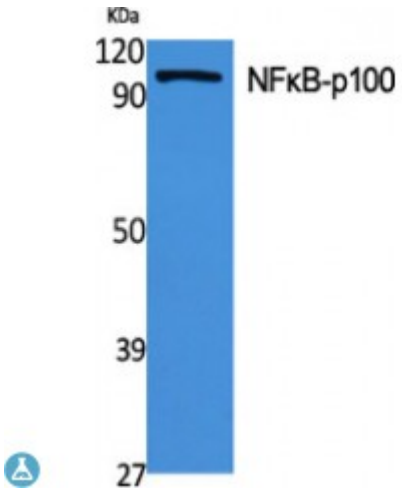
Anti-N kappa-p100 antibody



Description	Rabbit polyclonal to NFkappaB-p100.

Model	STJ94458
Host	Rabbit
Reactivity	Human, Mouse, Rat
Applications	ELISA, IF, IHC, IP, WB
Immunogen	Synthesized peptide derived from human NFkappaB-p100 around the non-phosphorylation site of S865.
Immunogen Region	810-890 aa
Gene ID	4791
Gene Symbol	NFKB2
Dilution range	WB 1:500-1:2000IHC 1:100-1:300IP 1:200-500IF 1:200-1:1000ELISA 1:20000
Specificity	NFkappaB-p100 Polyclonal Antibody detects endogenous levels of NFkappaB-p100 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 p100 subunit DNA-binding factor KBF2 H2TF1 Lymphocyte translocation chromosome 10 protein Nuclear factor of kappa light polypeptide gene enhancer in B-cells 2 Oncogene Lyt-10 Lyt10 Nuclea
Molecular Weight	97 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:7795OMIM:164012
Alternative Names	Nuclear factor NF-kappa-B p100 subunit DNA-binding factor KBF2 H2TF1 Lymphocyte translocation chromosome 10 protein Nuclear factor of kappa light polypeptide gene enhancer in B-cells 2 Oncogene Lyt-10 Lyt10 Nuclea
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. 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. In a non-canonical activation pathway, the MAP3K14-activated CHUK/IKKA homodimer phosphorylates NFKB2/p100 associated with RelB, inducing its proteolytic processing to NFKB2/p52 and the formation of NF-kappa-B RelB-p52 complexes. The NF-kappa-B heterodimeric RelB-p52 complex is a transcriptional activator. The NF-kappa-B p52-p52 homodimer is a transcriptional repressor. NFKB2 appears to have dual functions such as cytoplasmic retention of attached NF-kappa-B proteins by p100 and generation of p52 by a cotranslational processing. The proteasome-mediated process ensures the production of both p52 and p100 and preserves their independent function. p52 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. p52 and p100 are respectively the minor and major form; the processing of p100 being relatively poor. Isoform p49 is a subunit of the NF-kappa-B protein complex, which stimulates the HIV enhancer in synergy with p65. In concert with RELB, regulates the circadian clock by repressing the transcriptional activator activity of the CLOCK-ARNTL/BMAL1 heterodimer.
Sequence and Domain Family	The C-terminus of p100 might be involved in cytoplasmic retention, inhibition of DNA-binding by p52 homodimers, and/or transcription activation. The glycine-rich region (GRR) appears to be a critical element in the generation of

	p52.
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 p52 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.; Subsequent to MAP3K14-dependent serine phosphorylation, p100 polyubiquitination occurs then triggering its proteasome-dependent processing. Constitutive processing is tightly suppressed by its C-terminal processing inhibitory domain, named PID, which contains the death domain.