

# NFκB-p105 (phospho Ser927) Polyclonal Antibody

Catalog # AP67794

## Product Information

Application	WB, IHC-P, IF, ICC, E
Primary Accession	<a href="#">P19838</a>
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	105356

## Additional Information

Gene ID	4790
Other Names	NFKB1; 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
Dilution	WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/20000. Not yet tested in other applications. IHC-P~~N/A IF~~1:50~200 ICC~~N/A E~~N/A
Format	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.
Storage Conditions	-20°C

## Protein Information

Name	NFKB1
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.

#### Cellular Location

[Nuclear factor NF- $\kappa$ B p105 subunit]: Cytoplasm

## Background

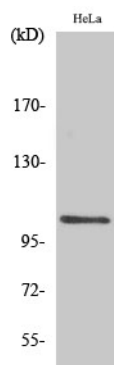
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NF- $\kappa$ B is a pleiotropic transcription factor present in almost all cell types and is the endpoint of a series of signal transduction events that are initiated by a vast array of stimuli related to many biological processes such as inflammation, 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.

## Images

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Western Blot analysis of various cells using  
Phospho-NF $\kappa$ B-p105 (S927) Polyclonal Antibody



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