

**Anti-NF-kappaB p105 (pS903) Antibody**  
**Rabbit polyclonal antibody to NF-kappaB p105 (pS903)**  
**Catalog # AP61144**

**Specification**

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**Anti-NF-kappaB p105 (pS903) Antibody - Product Information**

Application	WB
Primary Accession	<a href="#">P19838</a>
Reactivity	Human, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	105356

**Anti-NF-kappaB p105 (pS903) Antibody - Additional Information**

**Gene ID** 4790

**Other 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

**Target/Specificity**

Recognizes endogenous levels of NF-kappaB p105 (pS903) protein.

**Dilution**

WB~~WB (1/500 - 1/1000)

**Format**

Liquid in 0.42% Potassium phosphate, 0.87% Sodium chloride, pH 7.3, 30% glycerol, and 0.09% (W/V) sodium azide.

**Storage**

Store at -20 °C.Stable for 12 months from date of receipt

**Anti-NF-kappaB p105 (pS903) Antibody - 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

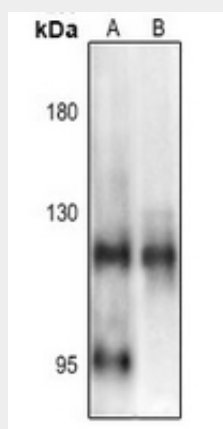
[Nuclear factor NF-kappa-B p105 subunit]: Cytoplasm

### Anti-NF-kappaB p105 (pS903) Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### Anti-NF-kappaB p105 (pS903) Antibody - Images



Western blot analysis of NF-kappaB p105 (pS903) expression in MCF7-TNFα (A), rat spleen (B) whole cell lysates.

### Anti-NF-kappaB p105 (pS903) Antibody - Background

KLH-conjugated synthetic peptide encompassing a sequence within the C-term region of human

NF-kappaB p105 (pS903). The exact sequence is proprietary.