

Phospho-NF kappa B(S536) Antibody
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP3749a**Specification**

Phospho-NF kappa B(S536) Antibody - Product Information

Application	DB,E
Primary Accession	Q04206
Other Accession	Q04207 , NP_068810.3 , NP_001138610.1 , Q7TON4
Reactivity	Human
Predicted	Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	60219

Phospho-NF kappa B(S536) Antibody - Additional Information**Gene ID** 5970**Other Names**

Transcription factor p65, Nuclear factor NF-kappa-B p65 subunit, Nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, RELA, NFKB3

Target/Specificity

This NF kappa B Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding S536 of human NF kappa B.

Dilution

DB~~1:500

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

Phospho-NF kappa B(S536) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Phospho-NF kappa B(S536) Antibody - Protein Information**Name** RELA

Synonyms NFKB3

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 heterodimeric RELA-NFKB1 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. The NF-kappa-B heterodimeric RELA-NFKB1 and RELA-REL complexes, for instance, function as transcriptional activators. 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. The inhibitory effect of I- kappa-B on NF-kappa-B through retention in the cytoplasm is exerted primarily through the interaction with RELA. RELA shows a weak DNA- binding site which could contribute directly to DNA binding in the NF- kappa-B complex. Besides its activity as a direct transcriptional activator, it is also able to modulate promoters accessibility to transcription factors and thereby indirectly regulate gene expression. Associates with chromatin at the NF-kappa-B promoter region via association with DDX1. Essential for cytokine gene expression in T- cells (PubMed:[15790681](#)). The NF-kappa-B homodimeric RELA-RELA complex appears to be involved in invasin-mediated activation of IL-8 expression. Key transcription factor regulating the IFN response during SARS-CoV-2 infection (PubMed:[33440148](#)).

Cellular Location

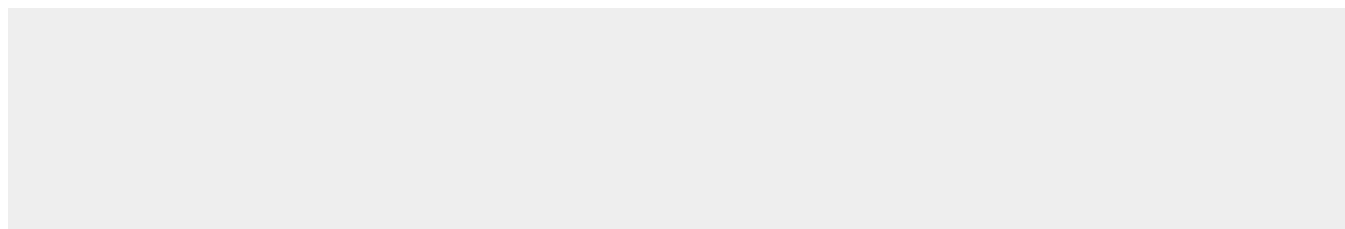
Nucleus. Cytoplasm. Note=Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I-kappa-B) (PubMed:1493333). Colocalized with DDX1 in the nucleus upon TNF-alpha induction (PubMed:19058135). Colocalizes with GFI1 in the nucleus after LPS stimulation (PubMed:20547752). Translocation to the nucleus is impaired in L.monocytogenes infection (PubMed:20855622)

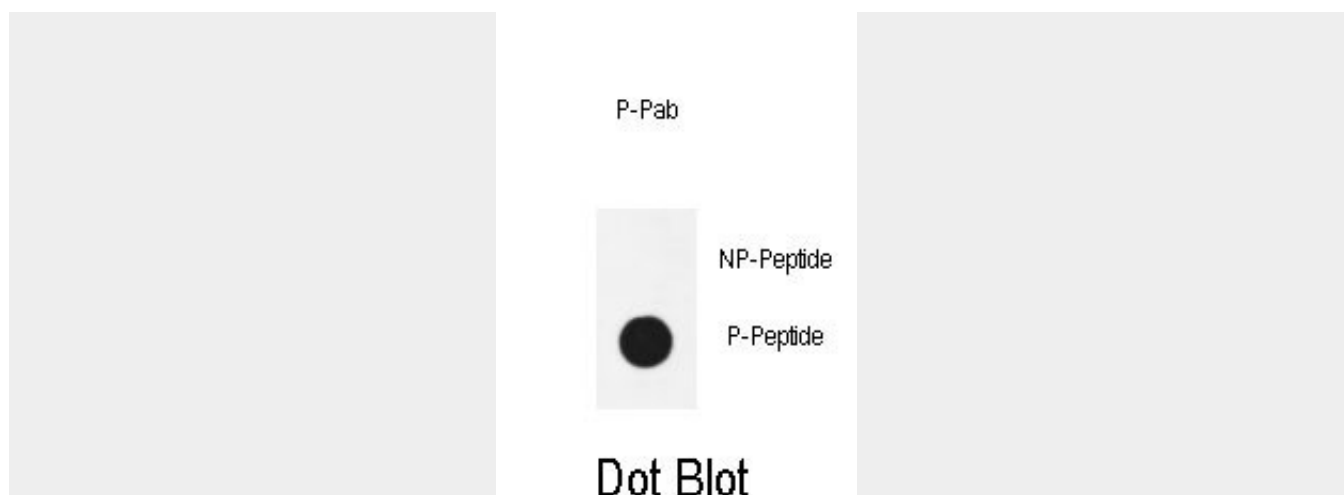
Phospho-NF kappa B(S536) Antibody - Protocols

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](#)

Phospho-NF kappa B(S536) Antibody - Images





Dot blot analysis of anti-Phospho-NF kappa B-S536 Phospho-specific Pab (Cat. #AP3749a) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibody working concentrations are 0.6ug per ml.

Phospho-NF kappa B(S536) Antibody - Background

NFKB1 (MIM 164011) or NFKB2 (MIM 164012) is bound to REL (MIM 164910), RELA, or RELB (MIM 604758) to form the NFKB complex. The p50 (NFKB1)/p65 (RELA) heterodimer is the most abundant form of NFKB. The NFKB complex is inhibited by I-kappa-B proteins (NFKBIA, MIM 164008 or NFKBIB, MIM 604495), which inactivate NFKB by trapping it in the cytoplasm. Phosphorylation of serine residues on the I-kappa-B proteins by kinases (IKBKA, MIM 600664, or IKBKB, MIM 603258) marks them for destruction via the ubiquitination pathway, thereby allowing activation of the NFKB complex. Activated NFKB complex translocates into the nucleus and binds DNA at kappa-B-binding motifs such as 5-prime GGGRNNYYCC 3-prime or 5-prime HGGARNYYCC 3-prime (where H is A, C, or T; R is an A or G purine; and Y is a C or T pyrimidine).

Phospho-NF kappa B(S536) Antibody - References

Pan, W.W., et al. J. Biol. Chem. 285(45):34348-34354(2010)
 Tago, K., et al. J. Biol. Chem. 285(40):30622-30633(2010)
 Park, J.S., et al. Oncol. Rep. 24(3):709-714(2010)
 Yu, Z.H., et al. Xi Bao Yu Fen Zi Mian Yi Xue Za Zhi 26(7):650-652(2010)
 Rohwer, N., et al. PLoS ONE 5 (8), E12038 (2010) :

Phospho-NF kappa B(S536) Antibody - Citations

- [Sea Cucumber Hydrolysate Alleviates Immunosuppression and Gut Microbiota Imbalance Induced by Cyclophosphamide in Balb/c Mice through the NF-κB Pathway](#)