

# Phospho-NFKBp65(S536) Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP3176a

## Product Information

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<b>Application</b>	WB, IHC-P, E
<b>Primary Accession</b>	<a href="#">Q04206</a>
<b>Other Accession</b>	<a href="#">Q04207</a> , <a href="#">Q6QN13</a> , <a href="#">P49895</a>
<b>Reactivity</b>	Human, Mouse
<b>Predicted</b>	Mouse, Pig
<b>Host</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>Isotype</b>	Rabbit IgG
<b>Calculated MW</b>	60219

## Additional Information

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<b>Gene ID</b>	5970
<b>Other Names</b>	Transcription factor p65, Nuclear factor NF-kappa-B p65 subunit, Nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, RELA, NFKB3
<b>Target/Specificity</b>	This NFKBp65 Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding S536 of human NFKBp65.
<b>Dilution</b>	WB~~1:1000 IHC-P~~1:100~500 E~~Use at an assay dependent concentration.
<b>Format</b>	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.
<b>Storage</b>	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.
<b>Precautions</b>	Phospho-NFKBp65(S536) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

## Protein Information

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<b>Name</b>	RELA
<b>Synonyms</b>	NFKB3
<b>Function</b>	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 invasion-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 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)

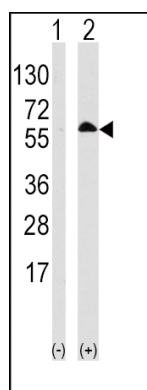
## Background

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).[supplied by OMIM].

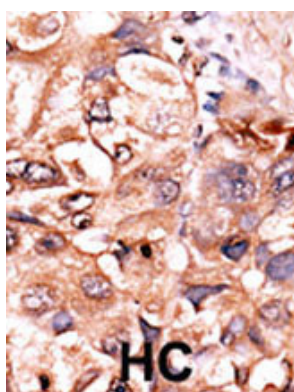
## References

- Li, Z., et al., J. Biol. Chem. 280(17):16843-16850 (2005).
- Su, H., et al., Science 307(5714):1465-1468 (2005).
- Burkhart, B.A., et al., J. Biol. Chem. 280(8):6349-6358 (2005).
- Cao, H.J., et al., World J. Gastroenterol. 11(6):903-907 (2005).
- Riggins, R.B., et al., Mol. Cancer Ther. 4(1):33-41 (2005).

## Images



Western blot analysis of Phospho-NFKBp65-S536 Antibody in human TNF alpha activated Hela cell line lysates. Phospho-RPS6KB1 (arrow) was detected using the purified PAb. (lane 1: without TNF alpha; lane 2: 10ng/ml TNF alpha, 5min)



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.