

**Anti-NFKB p65 (Rel A) pS536 (RABBIT) Antibody**  
**NFkB p65 phospho S536 Antibody**  
**Catalog # ASR5177****Specification**

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**Anti-NFKB p65 (Rel A) pS536 (RABBIT) Antibody - Product Information**

Host	Rabbit
Conjugate	Unconjugated
Target Species	Human
Reactivity	Human
Clonality	Polyclonal
Application	WB, IHC, E, I, LCI
Application Note	This affinity purified antibody has been tested for use in ELISA, immunohistochemistry and western blotting. Specific conditions for reactivity should be optimized by the end user. By western blot, a band approximately 65 kDa in size corresponding to phosphorylated p65 (RelA) protein is expected in the appropriate cell lysate or extract. This phospho-specific polyclonal antibody reacts with human p65 (RelA) pS536 and shows minimal reactivity by western blot with non-phosphorylated p65 (RelA) and minimal reactivity by ELISA against the non-phosphorylated form of the immunizing peptide.
Physical State	Liquid (sterile filtered)
Buffer	0.02 M Potassium Phosphate, 0.15 M Sodium Chloride, pH 7.2
Immunogen	This affinity purified antibody was prepared from whole rabbit serum produced by repeated immunizations with a synthetic peptide corresponding to a c-terminal region near phospho Serine S536 of human p65 (RelA) protein.
Preservative	0.01% (w/v) Sodium Azide

**Anti-NFKB p65 (Rel A) pS536 (RABBIT) Antibody - Additional Information****Gene ID** 5970**Other Names**  
5970**Purity**

This product was affinity purified from monospecific antiserum by immunoaffinity chromatography using phospho-peptide coupled to agarose beads followed by solid phase adsorption against nonphospho-peptide. This antibody is specific for human p65 (RelA) protein phosphorylated at

S536. A BLAST analysis was used to suggest cross reactivity with p65 (RelA) from human, mouse and rat based on 100% homology with the immunizing sequence. Cross reactivity with p65 (RelA) from other sources has not been determined.

#### Storage Condition

Store vial at -20° C prior to opening. Aliquot contents and freeze at -20° C or below for extended storage. Avoid cycles of freezing and thawing. Centrifuge product if not completely clear after standing at room temperature. This product is stable for several weeks at 4° C as an undiluted liquid. Dilute only prior to immediate use.

#### Precautions Note

This product is for research use only and is not intended for therapeutic or diagnostic applications.

### Anti-NFKB p65 (Rel A) pS536 (RABBIT) 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. Beside 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:<a href="http://www.uniprot.org/citations/15790681" target="\_blank">15790681</a>). 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:<a href="http://www.uniprot.org/citations/33440148" target="\_blank">33440148</a>).

#### Cellular Location

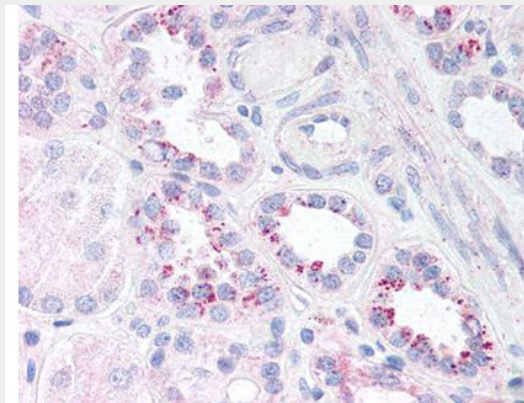
Nucleus. Cytoplasm. Note=Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I-kappa-B) (PubMed:1493333). Colocalized with DDX1 in the nucleus upon TNF-alpha induction (PubMed:19058135). Colocalizes with GFI1 in the nucleus after LPS stimulation (PubMed:20547752).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

## Anti-NFKB p65 (Rel A) pS536 (RABBIT) 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-NFKB p65 (Rel A) pS536 (RABBIT) Antibody - Images



Rockland's affinity purified anti-p65 (RelA) pS536 antibody was used at 5.0 µg/ml to detect signal in a variety of tissues including multi-human, multi-brain and multi-cancer slides. This image shows moderate positive staining of human kidney distal tubules and collecting ducts. Tissue was formalin-fixed and paraffin embedded. The image shows localization of the antibody as the precipitated red signal, with a hematoxylin purple nuclear counterstain. Personal Communication, Tina Roush, LifeSpanBiosciences, Seattle, WA.

## Anti-NFKB p65 (Rel A) pS536 (RABBIT) Antibody - Background

This antibody is designed, produced, and validated as part of a collaboration between Rockland and the National Cancer Institute (NCI) and is suitable for Cancer, Immunology and Nuclear Signaling research. NFκB was originally identified as a factor that binds to the immunoglobulin kappa light chain enhancer in B cells. It was subsequently found in non-B cells in an inactive cytoplasmic form consisting of NFκB bound to IκB. NFκB was originally identified as a heterodimeric DNA binding protein complex consisting of p65 (RelA) and p50 (NFκB1) subunits. Other identified subunits include p52 (NFκB2), cRel, and RelB. The p65, cRel, and RelB subunits are responsible for transactivation. The p50 and p52 subunits possess DNA binding activity but limited ability to transactivate. p52 has been reported to form transcriptionally active heterodimers with the NFκB subunit p65, similar to p50/p65 heterodimers. Low levels of p52 and p50 homodimers can also exist in cells. The heterodimers of p52/p65 and p50/p65 are regulated by physical inactivation in the cytoplasm by IκB-alpha. IκB-alpha binds to the p65 subunit, preventing nuclear localization, and DNA binding. Activators mediate a rapid phosphorylation of IκB by IκB kinase (IKK), which results in subsequent ubiquitination and proteolytic degradation. NFκB is then transported to the nucleus, where it activates transcription of target genes through binding to NFκB target sequences within the promoter. The HTLV-I protein Tax can induce constitutive NFκB activation through phosphorylation of both IκB-alpha and IκB-beta. The transforming protein Tax inhibits p53

transcriptional activity through the NF $\kappa$ B signaling pathway, specifically via the p65 (RelA) subunit. The inhibition of p53 activity is dependent upon phosphorylation of p65 (RelA) at S536 by the upstream kinase IKK $\beta$ .