

Anti-NFKB p65 (Rel A) pS276 (RABBIT) Antibody
NFkB p65 (RelA) Phospho S276 Antibody
Catalog # ASR3689

Specification

Anti-NFKB p65 (Rel A) pS276 (RABBIT) Antibody - Product Information

Host	Rabbit
Conjugate	Unconjugated
Target Species	Human
Reactivity	Human
Clonality	Polyclonal
Application	WB, IHC, E, I, LCI
Application Note	Phospho NFkB antibody reacts human pS276 p65 and shows minimal reactivity by western blot with non-phosphorylated p65 and minimal reactivity by ELISA against the non-phosphorylated form of the immunizing peptide. A 1:500 dilution has been used for staining p65 in human kidney tissue by IHC. Tissue was formalin fixed and paraffin embedded. Although not tested, this antibody is likely functional in immunoprecipitation. All conditions must be user optimized.
Physical State	Liquid (sterile filtered)
Immunogen	NFkB p65 (Rel A) peptide corresponding to an internal region near phospho Serine 276 of the human protein conjugated to Keyhole Limpet Hemocyanin (KLH).
Preservative	0.01% (w/v) Sodium Azide

Anti-NFKB p65 (Rel A) pS276 (RABBIT) Antibody - Additional Information

Gene ID 5970

Other Names 5970

Purity

Anti-NFkB p65 (RelA) Phospho S276 Antibody was prepared from monospecific antiserum by delipidation and defibrillation. This phospho specific polyclonal antibody is specific for phosphorylated pS276 human p65. Reactivity with non-phosphorylated p65 is minimal. Cross reactivity with pS276 phosphorylated p65 from mouse, rat or other species 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) pS276 (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. 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 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)

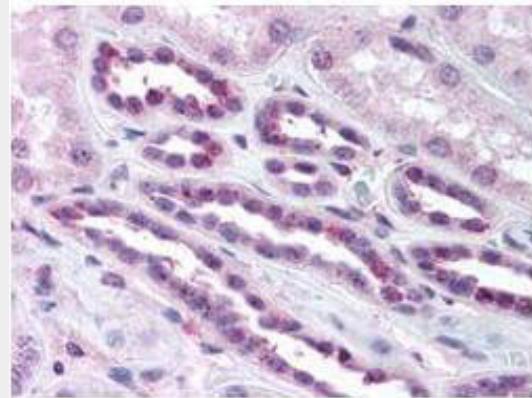
Anti-NFKB p65 (Rel A) pS276 (RABBIT) Antibody - Protocols

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) pS276 (RABBIT) Antibody - Images



Rockland's anti-p65 pS276 antibody was diluted 1:500 to detect p65 in human kidney tissue. Tissue was formalin fixed and paraffin embedded. No pre-treatment of sample was required. The image shows the localization of antibody as the precipitated red signal, with a hematoxylin purple nuclear counter stain.

Anti-NFKB p65 (Rel A) pS276 (RABBIT) Antibody - Background

NF- κ B 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 NF- κ B bound to I κ B. NF- κ B was originally identified as a heterodimeric DNA binding protein complex consisting of p65 (RelA) and p50 (NFKB1) subunits. Other identified subunits include p52 (NFKB2), 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 NF- κ B 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 I κ B-alpha. I κ B-alpha binds to the p65 subunit preventing nuclear localization and DNA binding. Activators mediate a rapid phosphorylation of I κ B by I κ B kinase (IKK) which results in subsequent ubiquitination and proteolytic degradation. NF- κ B is then transported to the nucleus, where it activates transcription of target genes through binding to NF- κ B target sequences within the promoter. The HTLV-1 protein Tax can induce constitutive NF- κ B activation through phosphorylation of both I κ B-alpha and I κ B-beta. The transforming protein Tax inhibits p53 transcriptional activity through the NF κ 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 β . Anti-NFKB antibody is ideal for Cell Biology, Nuclear Signaling, Neuroscience and Signal Transduction Research.