

Anti-NFkB p65 (Rel A) (RABBIT) Antibody
NFkB p65 Antibody
Catalog # ASR3801**Specification****Anti-NFkB p65 (Rel A) (RABBIT) Antibody - Product Information**

| | |
|------------------|--|
| Host | Rabbit |
| Conjugate | Unconjugated |
| Target Species | Human |
| Reactivity | Human, Mouse |
| Clonality | Polyclonal |
| Application | WB, IHC, E, IP, I, LCI |
| Application Note | Anti-NFkB p65 (Rel A) is tested by immunoblot of human and mouse NFkB p65 (Rel A), immunohistochemistry, and ELISA. This product was also tested in a gel supershift assay, IP, IF, and ChIP and found to be reactive against all p65 (Rel A) containing human, mouse or rat NFkB complexes using 0.5 to 1.0 µl per assay. |
| Physical State | Liquid (sterile filtered) |
| Immunogen | NFkB p65 (Rel A) peptide corresponding to a region near the C-terminus of the human protein conjugated to Keyhole Limpet Hemocyanin (KLH). |
| Preservative | 0.01% (w/v) Sodium Azide |

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

NFkB p65 (Rel A) was prepared from monospecific antiserum by delipidation and defibrination. Anti-NFkB p65 (Rel A) may react non-specifically with other proteins. Control peptide (code #100-4165p) will compete only with the specific reaction of antiserum with the NFkB p65 (Rel A) subunit.

Storage Condition

Store NF-kappaB antibody 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) (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: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

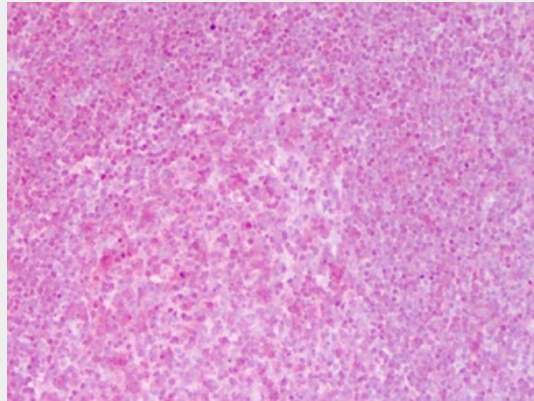
Nucleus. Cytoplasm. Note=Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I-kappa-B) (PubMed:1493333). Colocalized with DDX1 in the nucleus upon TNF-alpha induction (PubMed:19058135). Colocalizes with GFI1 in the nucleus after LPS stimulation (PubMed:20547752).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

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



Immunohistochemistry of NFKB p65 (Rel A) antibody. Tissue: lymphocytes and germinal center cells of the human tonsil. Fixation: formalin fixed paraffin embedded. Antigen retrieval: user optimized. Primary antibody: NFKB p65 (Rel A) antibody at 1:400. Secondary antibody: Peroxidase goat anti-rabbit at (p/n 611-103-122) 1:10,000 for 45 min at RT. Localization: nuclear and occasionally cytoplasmic. Staining: Moderate positive nuclear or cytoplasmic staining was observed in lymphocytes and germinal center cells of the tonsil.

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

Anti-NFkB p65 Antibody recognizes NFKB p65 which is a component of NFKB. NFKB 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 NFkappaB bound to IkappaB. NFkappaB was originally identified as a heterodimeric DNA binding protein complex consisting of p65 (RelA) and p50 (NFKB1) subunits. Other identified subunits include p52 (NFKB2), c-Rel, 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 NFkappaB subunit p65, similar to p50/p65 heterodimers. The heterodimers of p52/p65 and p50/p65 are regulated by physical inactivation in the cytoplasm by IkappaBalpha. IkappaBalpha binds to the p65 subunit, preventing nuclear localization and DNA binding. Low levels of p52 and p50 homodimers can also exist in cells.