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Anti-NFKB p65 (Rel A) N-TERMINAL SPECIFIC (RABBIT) Antibody

NFkB p65 Antibody
Catalog # ASR3802

Specification

Anti-NFKB p65 (Rel A) N-TERMINAL SPECIFIC (RABBIT) Antibody - Product Information

Host	**Rabbit**
Conjugate	**Unconjugated**
Target Species	**Human**
Reactivity	**Human**
Clonality	**Polyclonal**
Application	**WB, E, I, LCI**
Application Note	**Anti-NFkB p65 (N-terminal specific) has been tested for the detection of human NFkB p65 (N-terminal specific) by immunoblot. Functionality in supershift assays has not been determined.**
Physical State	**Liquid (sterile filtered)**
Immunogen	**Anti-NFkB p65 Antibody was produced by repeated immunizations with a synthetic NFkB p65 peptide corresponding to a region near the N-terminus of the human protein conjugated to Keyhole Limpet Hemocyanin (KLH).**
Preservative	**0.01% (w/v) Sodium Azide**

Anti-NFKB p65 (Rel A) N-TERMINAL SPECIFIC (RABBIT) Antibody - Additional Information

Gene ID 5970

Other Names
5970

Purity
NFkB p65 Antibody was prepared from monospecific antiserum by delipidation and defibrination. Anti-NFkB p65 (N-terminal specific) may react non-specifically with other proteins.

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) N-TERMINAL SPECIFIC (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-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) N-TERMINAL SPECIFIC (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 Cytomety
- Cell Culture

Anti-NFKB p65 (Rel A) N-TERMINAL SPECIFIC (RABBIT) Antibody - Images

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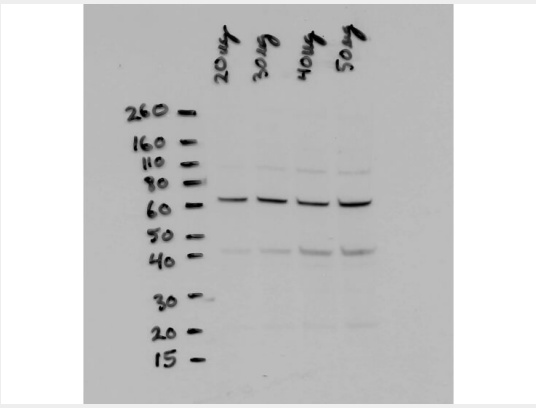

Western Blot of Rabbit anti-NFKB p65 N term antibody. Lane 1-4: PC3 cell lysate (p/n W09-001-GV6). Load: 20, 30, 40, and 50 ug per lane. Primary antibody: NFKB p65 antibody at 1:5000 for overnight at 4°C. Secondary antibody: HRP rabbit secondary antibody at 1:10,000 for 45 min at RT. Block: 5% BLOTTO overnight at 4°C. Predicted/Observed size: ~65kDa for NFKB p65. Other band(s): ~110kDa and ~45kDa.

Anti-NFKB p65 (Rel A) N-TERMINAL SPECIFIC (RABBIT) Antibody - Background

Anti-NFkB p65 Antibody detects NfkB p65. NFkB-p65 a subunit of NF-kappa-B transcription complex, which plays a crucial role in inflammatory and immune responses. The inhibitory effect of I-kappa-B upon NF-kappa-B in the cytoplasm is exerted primarily through the interaction with p65. P65 shows a weak DNA-binding site which could contribute directly to DNA binding in the NF-kappa-B complex. There are five NFkB proteins in mammals (RelA/NFkB-p65, RelB, c-Rel, NF-_B1/NFkB-p105, and NF-_B2/NFkB-p100). They form a variety of homodimers and heterodimers, each of which activates its own characteristic set of genes. Three splice-variant isoforms have been identified. Anti-NFkB p65 Antibody is ideal for investigators involved in Cell Signaling, Immunology, Cancer, and Signal Transduction research.