

Anti-NFkB p65 (Rel A) (MOUSE) Monoclonal Antibody
NFkB p65 Antibody
Catalog # ASR4127**Specification**

Anti-NFkB p65 (Rel A) (MOUSE) Monoclonal Antibody - Product Information

Host	Mouse
Conjugate	Unconjugated
Target Species	Human
Reactivity	Human
Clonality	Monoclonal
Application	WB, IHC, E, I, LCI
Application Note	Anti-NF-kB p65 Antibody is a mouse monoclonal antibody directed against NFkB p65 (Rel A) and recognizes a 65 kD band by Western blot against HeLa whole cell lysate. Control peptide (100-4165p) is sold separately. This product tested in WB, ICC, IHC, and IF.
Physical State	Liquid (sterile filtered)
Buffer	0.02 M Potassium Phosphate, 0.15 M Sodium Chloride, pH 7.2
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) (MOUSE) Monoclonal Antibody - Additional Information**Gene ID** 5970**Other Names**
5970**Purity**

Anti-NF-kB p65 Antibody was purified from concentrated tissue culture supernate by Protein A chromatography and showed a single band by IEP (immuno-electrophoresis) when tested with anti-mouse antibody. Reactivity was confirmed by ELISA against peptide conjugated carrier protein and by Western blot against HeLa whole cell lysate.

Storage Condition

Store NF-kB p65 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) (MOUSE) Monoclonal 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)

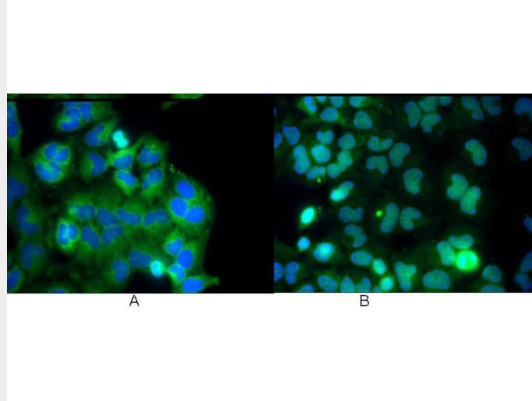
Anti-NFKB p65 (Rel A) (MOUSE) Monoclonal Antibody - Protocols

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) (MOUSE) Monoclonal Antibody - Images



Rockland Monoclonal anti NFKB p65 (Rel A) antibody was used to detect p65 by immunofluorescence at a dilution of 1:5000. HeLa cells were grown to sub-confluent on 18 mm² glass coverslips #1.5. Cells were either unstimulated (A), or stimulated (B) with 50 ng/ml of TNF alpha for 30 min prior fixation. Cells were then fixed in methanol and blocked with 10% normal goat serum (NGS), in PBS, and TritonX 0.2% (Tx) and incubated for 1 hr at RT with primary ab, counterstained with DAPI and washed in PBS/NGS/Tx. Cells were incubated for 1 hr at RT with Atto 425 conjugated anti mouse secondary antibody for STED CW imaging. Data was collected on a STED-CW TCS-SP5 Confocal system equipped with a DFC 350FX camera allowing sequential acquisition in widefield, confocal and STED CW imaging on the same system.

Anti-NFKB p65 (Rel A) (MOUSE) Monoclonal Antibody - Background

NFkappaB was originally identified as a factor that binds to the immunoglobulin kappa light chain enhancer in B cells. 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. Cell Biology, Nuclear Signaling, Neuroscience and Signal Transduction Research.