

Anti-NFkB p65 (Rel A) pS529 (RABBIT) Antibody
NFkB p65 (RelA) Phospho S529 Antibody
Catalog # ASR3690**Specification**

Anti-NFkB p65 (Rel A) pS529 (RABBIT) Antibody - Product Information

Host	Rabbit
Conjugate	Unconjugated
Target Species	Human
Reactivity	Human
Clonality	Polyclonal
Application	WB, IHC, E, I, LCI
Application Note	Anti-phospho NFkB antibody reacts human pS529 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. Although not tested, this antibody is likely functional in immunohistochemistry and immunoprecipitation.
Physical State	Liquid (sterile filtered)
Immunogen	NFkB p65 (Rel A) peptide corresponding to a region near phospho Serine 529 of the human protein conjugated to Keyhole Limpet Hemocyanin (KLH).
Preservative	0.1% (w/v) Sodium Azide

Anti-NFkB p65 (Rel A) pS529 (RABBIT) Antibody - Additional Information**Gene ID** 5970**Other Names**
5970**Purity**

Anti-NFkB p65 (RelA) Phospho S529 Antibody was prepared from monospecific antiserum by delipidation and defibrination. This phospho specific polyclonal antibody is specific for phosphorylated pS529 human p65. Reactivity with non-phosphorylated p65 is minimal. Cross reactivity with pS529 phosphorylated p65 from mouse, rat or other species has not been determined.

Storage Condition

Store NFkB p65 (RelA) Phospho S529 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) pS529 (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)

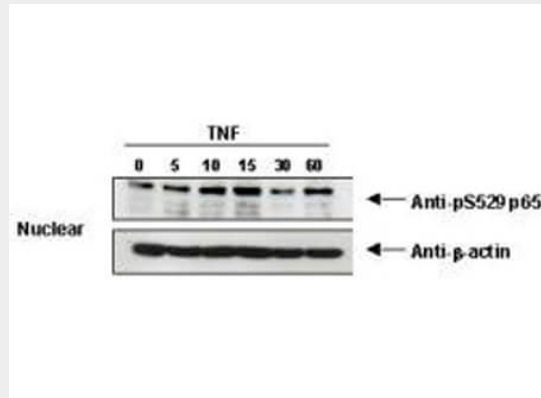
Anti-NFKB p65 (Rel A) pS529 (RABBIT) Antibody - Protocols

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) pS529 (RABBIT) Antibody - Images



TNF Induces phosphorylation of p65 in KBM-5 cells. Nuclear protein lysates prepared after 0, 5, 10, 15, 30 and 60 minutes of 0.1 nM TNF treatment of KBM-5 cells shows inducible phosphorylation using phospho specific polyclonal anti-human pS529 p65. Anti-beta-actin staining confirms loading of equivalent amounts of protein. HRP conjugated Gt-anti-Rabbit IgG was used to develop the blot using a chemiluminescent detection method. Other detection methods will yield similar results. Data contributed by Aggarwal BB, personal communication.

Anti-NFkB p65 (Rel A) pS529 (RABBIT) Antibody - Background

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 NFkB bound to IκB. NFkB 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 NFkB 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. NFkB is then transported to the nucleus, where it activates transcription of target genes through binding to NFkB target sequences within the promoter. The HTLV-I protein Tax can induce constitutive NFkB activation through phosphorylation of both IκB-alpha and IκB-beta. The transforming protein Tax inhibits p53 transcriptional activity through the NFkB signaling pathway, specifically via the p65 (RelA) subunit. Anti-NFkB antibody is ideal for Cell Biology, Nuclear Signaling, Neuroscience and Signal Transduction Research.