

NF-ĸB p65 Antibody

Rabbit mAb Catalog # AP90101

Product Information

| Application Primary Accession Reactivity Clonality Other Names | WB, IHC, IF, FC, ICC, IP, IHF <u>Q04206</u> Human, Mouse Monoclonal NFKB3; Transcription factor p65; p65; Nuclear factor NF-kappa-B p65 subunit, RELA; TF65; |
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| lsotype | Rabbit IgG |
| Host | Rabbit |
| Calculated MW | 60219 |

Additional Information

| Dilution Purification | WB 1:5000~1:10000 IHC 1:50~1:100 ICC/IF 1:50~1:100 |
|------------------------------|--|
| | Affinity-chromatography |
| Immunogen | A synthesized peptide derived from human NF-кВ p65 |
| Description | NFKB1 (MIM 164011) or NFKB2 (MIM 164012) is bound to REL (MIM 164910), |
| - | RELA, or RELB (MIM 604758) to form the NFKB complex. The p50 (NFKB1)/p65 |
| | (RELA) heterodimer is the most abundant form of NFKB. The NFKB complex is |
| | inhibited by I-kappa-B proteins (NFKBIA, MIM 164008 or NFKBIB, MIM |
| | 604495), which inactivate NFKB by trapping it in the cytoplasm. |
| Storage Condition and Buffer | Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. Store at +4°C short term. Store at -20°C long term. |
| | Avoid freeze / thaw cycle. |

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). |
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| 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) |

Images



| Image not found : 202311/AP90101-wb5.jpg | JLX001 Modulated the Inflammatory Reaction and Oxidative Stress in pMCAO Rats via Inhibiting the TLR2/4-NF-ĸB Signaling PathwayNeurochemical Research |
|--|--|
| Image not found : 202311/AP90101-wb6.jpg | Celecoxib protects hyperoxia-induced lung injury via NF-кB and AQP1Frontiers in pediatrics |

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