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Anti-RELA / NFKB p65 Antibody (clone 8G3)

Mouse Anti Human Monoclonal Antibody
Catalog # ALS18403

Specification

Anti-RELA / NFKB p65 Antibody (clone 8G3) - Product Information

Application	**WB, IHC-P, IF, E**
Primary Accession	Q04206
Predicted	**Human**
Host	**Mouse**
Clonality	**Monoclonal**
Isotype	**IgG1,l**
Calculated MW	**60219**
Dilution	**WB~~1:1000** **IHC-P~~N/A** **IF~~1:50~200** **E~~N/A**

Anti-RELA / NFKB p65 Antibody (clone 8G3) - Additional Information

Gene ID 5970

Alias Symbol	**RELA**

Other Names
RELA, NF-kappa-B p65delta3, NFKB3, p65, Transcription factor p65

Target/Specificity
Human RELA / NFKB p65

Reconstitution & Storage
Protein A purified

Precautions
Anti-RELA / NFKB p65 Antibody (clone 8G3) is for research use only and not for use in diagnostic or therapeutic procedures.

Anti-RELA / NFKB p65 Antibody (clone 8G3) - 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-RELA / NFKB p65 Antibody (clone 8G3) - 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-RELA / NFKB p65 Antibody (clone 8G3) - Images

Anti-RELA / NFKB p65 Antibody (clone 8G3) - Citations

- Euphorbia factor L2 alleviates lipopolysaccharide-induced acute lung injury and inflammation in mice through the suppression of NF-κB activation.