

NFκB-p105/p50 Polyclonal Antibody

Catalog # AP71284

Product Information

Application	WB, IHC-P, IF
Primary Accession	<u>P19838</u>
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	105356

Additional Information

Gene ID	4790
Other Names	NFKB1; Nuclear factor NF-kappa-B p105 subunit; DNA-binding factor KBF1; EBP-1; Nuclear factor of kappa light polypeptide gene enhancer in B-cells 1
Dilution	WB~~1:1000 IHC-P~~IF: 1:50-200 Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/20000. Not yet tested in other applications. IF~~IF: 1:50-200 Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/20000. Not yet tested in other applications.
Format	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.
Storage Conditions	-20°C

Protein Information

Name	NFKB1
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 and the heterodimeric p65-p50 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. 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. NF-kappa-B heterodimeric p65-p50 and RelB-p50 complexes are transcriptional activators. The NF-kappa-B p50-p50 homodimer is a transcriptional repressor, but can act as a transcriptional activator when associated with BCL3. NFKB1 appears to have dual functions such as cytoplasmic retention of attached NF-kappa-B proteins by p105 and generation of p50 by a cotranslational processing. The proteasome-mediated process ensures the production of both p50 and p105 and preserves their independent function, although processing of NFKB1/p105 also appears to occur post-translationally. p50 binds to the kappa-B consensus sequence 5'-GGRNNYYCC-3', located in the enhancer region of genes involved in immune response and acute phase reactions. In a complex with MAP3K8, NFKB1/p105 represses MAP3K8-induced MAPK signaling; active MAP3K8 is released by proteasome-dependent degradation of NFKB1/p105.

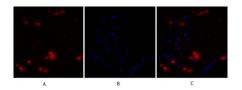
Cellular Location

[Nuclear factor NF-kappa-B p105 subunit]: Cytoplasm

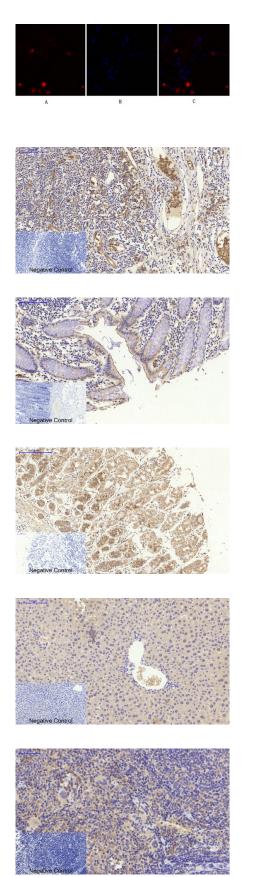
Background

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 and the heterodimeric p65-p50 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. 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. NF-kappa-B heterodimeric p65-p50 and RelB-p50 complexes are transcriptional activators. The NF-kappa-B p50-p50 homodimer is a transcriptional repressor, but can act as a transcriptional activator when associated with BCL3. NFKB1 appears to have dual functions such as cytoplasmic retention of attached NF-kappa-B proteins by p105 and generation of p50 by a cotranslational processing. The proteasome-mediated process ensures the production of both p50 and p105 and preserves their independent function, although processing of NFKB1/p105 also appears to occur post-translationally. p50 binds to the kappa-B consensus sequence 5'-GGRNNYYCC-3', located in the enhancer region of genes involved in immune response and acute phase reactions. In a complex with MAP3K8, NFKB1/p105 represses MAP3K8-induced MAPK signaling; active MAP3K8 is released by proteasome-dependent degradation of NFKB1/p105.

Images



Immunofluorescence analysis of human-lung tissue. 1,NFkB-p105/p50 Polyclonal Antibody(red) was diluted at 1:200(4°C,overnight). 2, Cy3 labled Secondary antibody was diluted at 1:300(room temperature, 50min).3, Picture B: DAPI(blue) 10min. Picture A:Target. Picture B: DAPI. Picture C: merge of A+B



Immunofluorescence analysis of human-lung tissue. 1,NFkB-p105/p50 Polyclonal Antibody(red) was diluted at 1:200(4°C,overnight). 2, Cy3 labled Secondary antibody was diluted at 1:300(room temperature, 50min).3, Picture B: DAPI(blue) 10min. Picture A:Target. Picture B: DAPI. Picture C: merge of A+B

Immunohistochemical analysis of paraffin-embedded Human-Tonsil tissue. 1,NFκB-p105/p50 Polyclonal Antibody was diluted at 1:200(4°C,overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval(>98°C,20min). 3,Secondary antibody was diluted at 1:200(room tempeRature, 30min). Negative control was used by secondary antibody only.

Immunohistochemical analysis of paraffin-embedded Human-colon tissue. 1,NFkB-p105/p50 Polyclonal Antibody was diluted at 1:200(4°C,overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval(>98°C,20min). 3,Secondary antibody was diluted at 1:200(room tempeRature, 30min). Negative control was used by secondary antibody only.

Immunohistochemical analysis of paraffin-embedded Human-stomach tissue. 1,NFκB-p105/p50 Polyclonal Antibody was diluted at 1:200(4°C,overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval(>98°C,20min). 3,Secondary antibody was diluted at 1:200(room tempeRature, 30min). Negative control was used by secondary antibody only.

Immunohistochemical analysis of paraffin-embedded Mouse-liver tissue. 1,NFκB-p105/p50 Polyclonal Antibody was diluted at 1:200(4°C,overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval(>98°C,20min). 3,Secondary antibody was diluted at 1:200(room tempeRature, 30min). Negative control was used by secondary antibody only.

Immunohistochemical analysis of paraffin-embedded Mouse-spleen tissue. 1,NFkB-p105/p50 Polyclonal Antibody was diluted at 1:200(4°C,overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval(>98°C,20min). 3,Secondary antibody was diluted at 1:200(room tempeRature, 30min). Negative control was used by secondary antibody only.

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