

NF- κ B p65 (Phospho Thr435) Rabbit pAb

CatalogNo: YP0193 **Orthogonal Validated** 

Comparable Abs 

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse, Rat, Monkey

Applications

- WB, IHC, IF, IP, ELISA

MW

- 60kD (Observed)

Isotype

- IgG

Storage

Storage* -15°C to -25°C/1 year (Do not lower than -25°C)

Formulation Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Recommended Dilution Ratios

WB 1:500-1:2000

IHC 1:100-1:300

IP 2-5 ug/mg lysate

ELISA 1:20000

IF 1:50-200

Basic Information

Clonality Polyclonal

Immunogen Information

Immunogen The antiserum was produced against synthesized peptide derived from human NF- κ B p65 around the phosphorylation site of Thr435. AA range:402-451

Specificity

Phospho-NFκB-p65 (T435) Polyclonal Antibody detects endogenous levels of NFκB-p65 protein only when phosphorylated at T435. The name of modified sites may be influenced by many factors, such as species (the modified site was not originally found in human samples) and the change of protein sequence (the previous protein sequence is incomplete, and the protein sequence may be prolonged with the development of protein sequencing technology). When naming, we will use the "numbers" in historical reference to keep the sites consistent with the reports. The antibody binds to the following modification sequence (lowercase letters are modification sites):EGtLS

| Target Information

Gene name RELA

Protein Name Transcription factor p65

Organism	Gene ID	UniProt ID
Human	5970;	Q04206;
Mouse	19697;	Q04207;

Cellular Localization

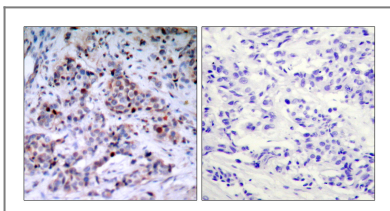
Nucleus . Cytoplasm . Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I-kappa-B) (PubMed:1493333). Colocalized with DDX1 in the nucleus upon TNF-alpha induction (PubMed:19058135). Colocalizes with GFI1 in the nucleus after LPS stimulation (PubMed:20547752). Translocation to the nucleus is impaired in L.monocytogenes infection (PubMed:20855622). .

Tissue specificity Bone,Colon,Pancreas,Placenta,

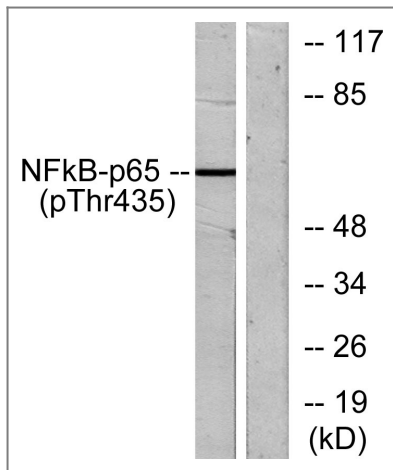
Function

Function: NF-kappa-B is a pleiotropic transcription factor which is present in almost all cell types and is involved in 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 p65-c-Rel complexes are transcriptional activators. The NF-kappa-B p65-p65 complex appears to be involved in invasion-mediated activation of IL-8 expression. The inhibitory effect of I-kappa-B upon NF-kappa-B in the cytoplasm is exerted primarily through the interaction with p65. p65 shows a weak DNA-binding site which could contribute directly to DNA binding in the NF-kappa-B complex. PTM: Phosphorylation on 'Ser-536' stimulates acetylation on 'Lys-310' and interaction with CBP; the phosphorylated and acetylated forms show enhanced transcriptional activity. PTM: Reversibly acetylated; the acetylation seems to be mediated by CBP, the deacetylation by HDAC3. Acetylation at 'Lys-122' enhances DNA binding and impairs association with NFKBIA. Acetylation at 'Lys-310' is required for full transcriptional activity in the absence of effects on DNA binding and NFKBIA association. Acetylation can also lower DNA-binding and results in nuclear export. PTM: Ubiquitinated, leading to its proteasomal degradation. Degradation is required for termination of NF-kappa-B response. Similarity: Contains 1 RHD (Rel-like) domain. Subcellular location: Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I-kappa-B). Subunit: Component of the NF-kappa-B p65-p50 complex. Component of the NF-kappa-B p65-c-Rel complex. Homodimer; component of the NF-kappa-B p65-p65 complex. Component of the NF-kappa-B p65-p52 complex. May interact with ETHE1. Binds AES and TLE1. Interacts with TP53BP2. Binds to and is phosphorylated by the activated form of either RPS6KA4 or RPS6KA5. Interacts with ING4 and this interaction may be indirect. Interacts with CARM1, USP48 and UNC5CL. Interacts with IRAK1BP1 (By similarity). Interacts with NFKBID (By similarity). Interacts with NFKBIA. Interacts with GSK3B. Interacts with NFKBIB (By similarity). Interacts with NFKBIE. Interacts with NFKBIZ (By similarity). Part of a 70-90 kDa complex at least consisting of CHUK, IKBKB, NFKBIA, RELA, IKBKAP and MAP3K14. Interacts with HDAC3; HDAC3 mediates the deacetylation of RELA. Interacts with HDAC1; the interaction requires non-phosphorylated RELA. Interacts with CBP; the interaction requires phosphorylated RELA. Interacts (phosphorylated at 'Thr-254') with PIN1; the interaction inhibits p65 binding to NFKBIA. Interacts with SOCS1. Interacts with UXT. Interacts with MTDH. Interacts with human respiratory syncytial virus (HRSV) protein M2-1.

Validation Data



Immunohistochemistry analysis of paraffin-embedded human breast carcinoma, using NF-kappaB p65 (Phospho-Thr435) Antibody. The picture on the right is blocked with the phospho peptide.



Western blot analysis of lysates from COS7 cells treated with TNF-alpha, using NF-kappaB p65 (Phospho-Thr435) Antibody. The lane on the right is blocked with the phospho peptide.

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