

Immunotag™ NF-κB p65 Polyclonal Antibody

Antibody Specification	
Catalog No.	ITT5770
Product Description	Immunotag™ NF-κB p65 Polyclonal Antibody
Size	50 µg, 100 µg
Conjugation	HRP, Biotin, FITC, Alexa Fluor® 350, Alexa Fluor® 405, Alexa Fluor® 488, Alexa Fluor® 555, Alexa Fluor® 647
IMPORTANT NOTE	This product is custom manufactured with a lead time of 3-4 weeks. Once in production, this item cannot be cancelled or returned and is not eligible for return.
Target Protein	NFκB p65
Clonality	Polyclonal
Storage/Stability	-20°C/1 year
Application	WB,ELISA
Recommended Dilution	WB 1:500-2000, ELISA 1:10000-20000
Concentration	1 mg/ml
Reactive Species	Human,Mouse,Rat
Host Species	Rabbit
Immunogen	Synthetic Peptide of NF-κB p65
Specificity	NF-κB p65 Polyclonal Antibody detects endogenous levels of NF-κB p65
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific resin.
Form	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Gene Name	RELA NFKB3
Accession No.	Q04206 Q04207
Alternate Names	v-rel reticuloendotheliosis viral oncogene homolog A (avian)

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Description	RELA proto-oncogene, NF-kB subunit(RELA) Homo sapiens NF-kappa-B is a ubiquitous transcription factor involved in a wide variety of biological processes. It is held in the cytoplasm in an inactive state by specific inhibitors. Upon degradation of these inhibitors, NF-kappa-B moves to the nucleus and activates transcription of specific genes. NF-kappa-B is composed of two subunits, p50 and p65, bound to either REL, RELA, or RELB. The most abundant form of NF-kappa-B is NFKB1 complexed with the p50 subunit. RELA. Four transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, May 2014]
Cell Pathway/Category	MAPK_ERK_Growth,MAPK_G_Protein,Chemokine,Apoptosis_Inhibition,Apoptosis_Mitochondrial,Apoptosis_Regulation,IL-1-like receptor,RIG-I-like receptor,Cytosolic DNA-sensing pathway,T_Cell_Receptor,B_Cell_Antigen,Neurotrophin,Adipocytokine,Epithelial cell signaling in Helicobacter pylori infection,Pathways in cancer,Pancreatic cancer,Prostate cancer,Chronic myeloid leukemia,Acute myeloid leukemia,Non-small cell lung cancer,
Protein Expression	Bone,Colon,Pancreas,Placenta,
Subcellular Localization	nuclear chromatin,nucleus,nucleoplasm,transcription factor complex,cytoplasm,cytosol,I-kappaB/NF-kappaB complex,
Protein Function	function:NF-kappa-B is a pleiotropic transcription factor which is present in almost all cell types and is involved in a wide variety of biological processes such as inflammation, immunity, differentiation, cell growth, tumorigenesis and apoptosis. It is a homo- or heterodimeric complex formed by the Rel-like domain-containing proteins RELA/p65, RELB, NFKB1/p50, REL and NFKB2/p52 and the heterodimeric p65-p50 complex appears to be most abundant. The individual dimers bind at kappa-B sites in the DNA of their target genes and the individual dimers have distinct preferences for binding sites such 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 including phosphorylation, ubiquitination, compartmentalization as well as by interactions with other cofactors or corepressors. NF-kappa-B complexed with I-kappa-B in the cytoplasm in an inactive state complexed with members of the NF-kappa-B inhibitor (I-kappa-B) family. Upon activation, I-kappa-B is phosphorylated by I-kappa-B kinases (IKKs) in response to different activating stimuli and is degraded thus liberating the active NF-kappa-B complex which translocates to the nucleus. NF-kappa-B complexes with p65 and p65-c-Rel complexes are transcriptional activators. The NF-kappa-B p65-p65 complex appears to be involved in the regulation of IL-8 mediated activation of IL-8 expression. The inhibitory effect of I-kappa-B upon NF-kappa-B the cytoplasmic complex is mediated through the interaction with p65. p65 shows a weak DNA-binding site which could contribute directly to the DNA binding of the 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 on 'Lys-310' seems to be mediated by CBP, the deacetylation by HDAC3. Acetylation at 'Lys-122' enhances DNA binding and NFKBIA association. Acetylation at 'Lys-310' is required for full transcriptional activity in the absence of CBP. Acetylation can also lower DNA-binding and results in nuclear export.,PTM:Ubiquitination of the RHD (Rel-like) domain.,subcellular location:Nuclear, but also found in the cytoplasm in an inactive form complexed with the inhibitor (I-kappa-B).,subunit:Component of the NF-kappa-B p65-p50 complex. Component of the NF-kappa-B p65-p50 complex. Homodimer; component of the NF-kappa-B p65-p65 complex. Component of the NF-kappa-B p65-p65 complex. Interacts with ETHE1. Binds AES and TLE1. Interacts with TP53BP2. Binds to and is phosphorylated by the p38 MAPK, either RPS6KA4 or RPS6KA5. Interacts with ING4 and this interaction may be indirect. Interacts with CAAP1. Interacts with UNC5CL. Interacts with IRAK1BP1 (By similarity).Interacts with NFKBID (By similarity). Interacts with NFKB1. Interacts with GSK3B. Interacts with NFKBIB (By similarity). Interacts with NFKBIE. Interacts with NFKBIZ (By similarity). Interacts with the complex at least consisting of CHUK, IKBKB, NFKBIA, RELA, IKBKAP and MAP3K14. Interacts with HDAC3. Interacts with HDAC3; deacetylation of RELA. Interacts with HDAC1; the interaction requires non-phosphorylated RELA. Interacts with HDAC2; the interaction requires phosphorylated RELA. Interacts (phosphorylated at 'Thr-254') with PIN1; the interaction requires phosphorylated RELA. Interacts with SOCS1. Interacts with UXT. Interacts with MTDH. Interacts with human cytomegalovirus (HRSV) protein M2-1.,

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Usage	For Research Use Only! Not for diagnostic or therapeutic procedures.
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