SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com

[Marker Primary Antibody]

Rabbit Anti-NFKB p65/FITC Conjugated antibody

Catalog Number: SL0465R-FITC

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	Anti-NFKB p65/FITC
Immunogen:	KLH conjugated synthetic peptide derived from human NFKBp65
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Applications:	Flow-Cyt=1:50-200 IF=1:50-200 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human,Mouse(predicted:Rat,Chicken,Dog,Pig,Cow,Horse,Rabbit,Zebrafish,Sheep)
Background:	background: NF-kappa-B is a ubiquitous transcription factor involved in several biological processes. It is held in the cytoplasm in an inactive state by specific inhibitors. Upon degradation of the inhibitor, NF-kappa-B moves to the nucleus and activates transcription of specific genes. NF-kappa-B is composed of NFKB1 or NFKB2 bound to either REL, RELA, or RELB. The most abundant form of NF-kappa-B is NFKB1 complexed with the product of this gene, RELA. Four transcript variants encoding different isoforms have been found for this gene. [provided by RefSeq, Sep 2011]. **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,

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	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 invasin-mediated activation of IL-8 expression. The inhibitory effect of I-kappa-B upon NF-kappa-B 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. Associates with chromatin at the NF-kappa-B promoter region via association with DDX1. **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. Interacts with NFKBID. Interacts with NFKBIA. Interacts with GSK3B. Interacts with NFKBIB. Interacts with NFKBIE. Interacts with NFKBIZ. Interacts with EHMT1 (via ANK repeats). 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

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	(phosphorylated at 'Thr-254') with PIN1; the interaction inhibits p65 binding to NFKBIA. Interacts with SOCS1. Interacts with UXT. Interacts with MTDH and PHF11. Interacts with ARRB2. Interacts with human respiratory syncytial virus (HRSV) protein M2-1. Interacts with NFKBIA (when phosphorylated), the interaction is direct; phosphorylated NFKBIA is part of a SCF(BTRC)-like complex lacking CUL1. Interacts with RNF25. Interacts (via C-terminus) with DDX1. Interacts with UFL1 and COMMD1. Interacts with BRMS1; this promotes deacetylation of 'Lys-310'. Interacts with NOTCH2. Directly interacts with MEN1; this interaction represses NFKB-mediated transactivation. Interacts with AKIP1, which promotes the phosphorylation and nuclear retention of RELA. Interacts (via the RHD) with GFI1; the interaction, after bacterial lipopolysaccharide (LPS) stimulation, inhibits the transcriptional activity by interfering with the DNA-binding activity to target gene promoter DNA. **Subcellular Location:** Nucleus. Cytoplasm. Note=Colocalized with DDX1 in the nucleus upon TNF-alpha induction. Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I-kappa-B). Colocalizes with GFI1 in the nucleus after LPS stimulation. **Post-translational modifications:** Ubiquitinated, leading to its proteasomal degradation. Degradation is required for termination of NF-kappa-B response. Monomethylated at Lys-310 by SETD6. Monomethylation at Lys-310 is recognized by the ANK repeats of EHMT1 and promotes the formation of repressed chromatin at target genes, leading to down-regulation of NF-kappa-B transcription factor activity. Phosphorylation at Ser-311 disrupts the interaction with EHMT1 without preventing monomethylation at Lys-310 and relieves the repression of target genes. Phosphorylation at Ser-311 disrupts the interaction with EHMT1 and promotes transcription factor activity. Phosphorylation on Ser-536 stimulates acetylation on Lys-310 and interaction with CBP; the phosphorylated and acetylated forms show enhanced transcriptional activity. Phosphorylation at Ser-276 by RPS6KA4 and RPS6KA5 promotes its transactivation and transcriptional activities. Reversibly acetylated; the acetylation seems to be mediated by CBP, the deacetylation by HDAC3 and SIRT2. 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. Interaction with BRMS1 promotes deacetylation of Lys-310. Lys-310 is deacetylated by SIRT2.

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	S-nitrosylation of Cys-38 inactivates the enzyme activity. Sulfhydration at Cys-38 mediates the anti-apoptotic activity by promoting the interaction with RPS3 and activating the transcription factor activity. Sumoylation by PIAS3 negatively regulates DNA-bound activated NF-kappa-B. **Similarity:** Contains 1 RHD (Rel-like) domain. **Database links:** **Entrez Gene:** 5970 Human **Entrez Gene:** 19697 Mouse **Entrez Gene:** 309165 Rat Omim: 164014 Human **SwissProt:** Q04206 Human **SwissProt:** Q04207 Mouse **Unigene:** 502875 Human **Unigene:** 249966 Mouse **Unigene:** 19480 Rat Important **Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications. transcriptional regulatory factor（Transcriptin Regulators） NF-κB p65 is an important transcription factor. NF-κB p65 can activate the regulation of genes involved in inflammation, cell proliferation, Apoptosis, etc., affecting cell apoptosis and also affecting the sensitivity of Tumour cells to cytotoxic drugs and ionizing radiation. The oncogenic mutation induced by the ras gene requires activation of NFkB, suggesting that NFkB may play a role in carcinogenesis; other literature reports that NFkB is activated or expressed in human Tumours such as breast cancer, non-small cell lung cancer, thyroid cancer, T or Blymphocyte leukemia, and virus-induced Tumours. Research suggests that: NFKBp65 protein exists in the cytoplasm in a bound state under resting conditions, and when NFKBp65 protein is activated, it dissociates and enters the nucleus. (NF-κB and its inhibitory protein IκB family members bind, exists in the cytoplasm as an inactive complex, when cells are subjected to various stimuli, NF-κB dissociates from IκB, thereby entering the nucleus, binding to the corresponding target sequences, and regulating gene expression) NF-κB can protect cells from apoptosis caused by Tumour necrosis factor and ionizing radiation, while inhibiting NFkB expression can increase Apoptosis caused by TNF, etc., and increase the sensitivity of Tumour cells to chemotherapy and radiotherapy.