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NFKB(p100) Blocking Peptide (C-term S866/870)

Synthetic peptide
Catalog # BP19753B

Specification

NFKB(p100) Blocking Peptide (C-term S866/870) - Product Information

Primary Accession	Q00653
Other Accession	Q9WTK5, NP_001070961.1

NFKB(p100) Blocking Peptide (C-term S866/870) - Additional Information

Gene ID 4791

Other Names
Nuclear factor NF-kappa-B p100 subunit, DNA-binding factor KBF2, H2TF1, Lymphocyte translocation chromosome 10 protein, Nuclear factor of kappa light polypeptide gene enhancer in B-cells 2, Oncogene Lyt-10, Lyt10, Nuclear factor NF-kappa-B p52 subunit, NFKB2, LYT10

Target/Specificity
The synthetic peptide sequence is selected from aa 863-874 of HUMAN NFKB2

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

NFKB(p100) Blocking Peptide (C-term S866/870) - Protein Information

Name NFKB2

Synonyms LYT10

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 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. In a non-canonical activation pathway, the MAP3K14-activated CHUK/IKKA homodimer phosphorylates NFKB2/p100 associated with RelB, inducing its proteolytic processing to NFKB2/p52 and the formation of NF-kappa-B RelB-p52 complexes. The NF-kappa-B heterodimeric RelB-p52 complex is a transcriptional activator. The NF-kappa-B p52-p52 homodimer is a transcriptional repressor. NFKB2 appears to have dual functions such as cytoplasmic retention of attached NF-kappa-B proteins by p100 and generation of p52 by a cotranslational processing. The proteasome- mediated process ensures the production of both p52 and p100 and preserves their independent function. p52 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. p52 and p100 are respectively the minor and major form; the processing of p100 being relatively poor. Isoform p49 is a subunit of the NF-kappa-B protein complex, which stimulates the HIV enhancer in synergy with p65. In concert with RELB, regulates the circadian clock by repressing the transcriptional activator activity of the CLOCK-BMAL1 heterodimer.

Cellular Location
Nucleus. Cytoplasm. Note=Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I- kappa-B)

NFKB(p100) Blocking Peptide (C-term S866/870) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

NFKB(p100) Blocking Peptide (C-term S866/870) - Images

NFKB(p100) Blocking Peptide (C-term S866/870) - Background

NF-kappa-B has been detected in numerous cell types that express cytokines, chemokines, growth factors, cell adhesion molecules, and some acute phase proteins in health and in various disease states. NF-kappa-B is activated by a wide variety of stimuli, such as cytokines, oxidant-free radicals, inhaled particles, ultraviolet irradiation, and bacterial or viral products. Inappropriate activation of NF-kappa-B has been linked to inflammatory events associated with autoimmune arthritis, asthma, septic shock, lung fibrosis, glomerulonephritis, atherosclerosis, and AIDS. In contrast, complete and persistent inhibition of NF-kappa-B has been linked directly to apoptosis, inappropriate immune cell development, and delayed cell growth. NFKB1 (MIM 164011) and NFKB2 encode p105 and p100 proteins that are processed to produce the active p50 and p52 NF-kappa-B subunits, respectively. However, the p100 and p105 proteins serve regulatory functions and should not be considered exclusively as precursor forms. The most abundant activated form of NF-kappa-B is a heterodimer of the p50 or p52 subunit bound to the RELA subunit (MIM 164014). Other NF-kappa-B complexes, consisting of hetero- and homodimers of p50, p52, RELA, REL (MIM 164910), and RELB (MIM 604758), have also been detected. NF-kappa-B complexes are inhibited by I-kappa-B proteins, NFKBIA (MIM 164008) or NFKBIB (MIM 604495), which inactivate NF-kappa-B by trapping it in the cytoplasm. Phosphorylation of serine residues on the I-kappa-B

proteins by the kinases IKBKA (CHUK; MIM 600664) or IKBKB (MIM 603258) marks them for destruction via the ubiquitination pathway, thereby allowing activation of the NF-kappa-B complex. The activated NF-kappa-B complex translocates into the nucleus and binds DNA at kappa-B-binding motifs, such as 5-prime GGGRNNYYCC 3-prime or 5-prime HGGARNYYCC 3-prime (where H is A, C, or T; R is an A or G purine; and Y is a C or T pyrimidine). For reviews, see Chen et al. (1999) [PubMed 9895331] and Baldwin (1996) [PubMed 8717528].

NFKB(p100) Blocking Peptide (C-term S866/870) - References

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Johnatty, S.E., et al. PLoS Genet. 6 (7), E1001016 (2010) :
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