

## 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 <u>Q00653</u> Other Accession <u>Q9WTK5</u>,

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

# 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



### Synonyms LYT10

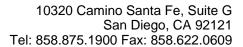
#### **Function**

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

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

Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)
Potter, C., et al. Ann. Rheum. Dis. 69(7):1315-1320(2010)
Schuurhof, A., et al. Pediatr. Pulmonol. 45(6):608-613(2010)
Johnatty, S.E., et al. PLoS Genet. 6 (7), E1001016 (2010):
Keller, U., et al. BMC Cancer 10, 348 (2010):





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-ARNTL/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