

**RELA Blocking Peptide (Center)**  
**Synthetic peptide**  
**Catalog # BP19822c****Specification**

---

**RELA Blocking Peptide (Center) - Product Information**

Primary Accession [Q04206](#)  
Other Accession [Q04207](#), [NP\\_001138610.1](#)

**RELA Blocking Peptide (Center) - Additional Information**

**Gene ID** 5970

**Other Names**

Transcription factor p65, Nuclear factor NF-kappa-B p65 subunit, Nuclear factor of kappa light polypeptide gene enhancer in B-cells 3, RELA, NFKB3

**Target/Specificity**

The synthetic peptide sequence is selected from aa 181-195 of HUMAN RELA

**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.

**RELA Blocking Peptide (Center) - Protein Information**

**Name** RELA

**Synonyms** NFKB3

**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 heterodimeric RELA-NFKB1 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. The NF-kappa-B heterodimeric RELA-NFKB1 and RELA-REL complexes, for instance, function as transcriptional activators. 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. The inhibitory effect of I- kappa-B on NF-kappa-B through retention in the cytoplasm is exerted primarily through the interaction with RELA. RELA shows a weak DNA- binding site which could contribute directly to DNA binding in the NF- kappa-B complex. Beside its activity as a direct transcriptional activator, it is also able to modulate promoters accessibility to transcription factors and thereby indirectly regulate gene expression. Associates with chromatin at the NF-kappa-B promoter region via association with DDX1. Essential for cytokine gene expression in T- cells (PubMed:<a href="http://www.uniprot.org/citations/15790681" target="\_blank">15790681</a>). The NF-kappa-B homodimeric RELA-RELA complex appears to be involved in invasin-mediated activation of IL-8 expression. Key transcription factor regulating the IFN response during SARS-CoV-2 infection (PubMed:<a href="http://www.uniprot.org/citations/33440148" target="\_blank">33440148</a>).

### **Cellular Location**

Nucleus. Cytoplasm. Note=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)

### **RELA Blocking Peptide (Center) - Protocols**

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

- [Blocking Peptides](#)

### **RELA Blocking Peptide (Center) - Images**

### **RELA Blocking Peptide (Center) - Background**

NFKB1 (MIM 164011) or NFKB2 (MIM 164012) is bound to REL (MIM 164910), RELA, or RELB (MIM 604758) to form the NFKB complex. The p50 (NFKB1)/p65 (RELA) heterodimer is the most abundant form of NFKB. The NFKB complex is inhibited by I-kappa-B proteins (NFKBIA, MIM 164008 or NFKBIB, MIM 604495), which inactivate NFKB by trapping it in the cytoplasm. Phosphorylation of serine residues on the I-kappa-B proteins by kinases (IKBKA, MIM 600664, or IKBKB, MIM 603258) marks them for destruction via the ubiquitination pathway, thereby allowing activation of the NFKB complex. Activated NFKB 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).

### **RELA Blocking Peptide (Center) - References**

Pan, W.W., et al. J. Biol. Chem. 285(45):34348-34354(2010)  
Tago, K., et al. J. Biol. Chem. 285(40):30622-30633(2010)  
Park, J.S., et al. Oncol. Rep. 24(3):709-714(2010)  
Yu, Z.H., et al. Xi Bao Yu Fen Zi Mian Yi Xue Za Zhi 26(7):650-652(2010)  
Rohwer, N., et al. PLoS ONE 5 (8), E12038 (2010) :