

**Phospho-IKKB(Y609) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP3886a**

**Specification**

---

**Phospho-IKKB(Y609) Blocking Peptide - Product Information**

Primary Accession  
Other Accession

[O14920](#)  
[O88351](#), [Q95KV0](#), [NP\\_001177649.1](#)

**Phospho-IKKB(Y609) Blocking Peptide - Additional Information**

**Gene ID** 3551

**Other Names**

Inhibitor of nuclear factor kappa-B kinase subunit beta, I-kappa-B-kinase beta, IKK-B, IKK-beta, IKBKB, I-kappa-B kinase 2, IKK2, Nuclear factor NF-kappa-B inhibitor kinase beta, NFKBIKB, IKBKB, IKKB

**Target/Specificity**

The synthetic peptide sequence is selected from aa 602-615 of HUMAN IKBKB

**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.

**Phospho-IKKB(Y609) Blocking Peptide - Protein Information**

**Name** IKBKB

**Synonyms** IKKB

**Function**

Serine kinase that plays an essential role in the NF-kappa-B signaling pathway which is activated by multiple stimuli such as inflammatory cytokines, bacterial or viral products, DNA damages or other cellular stresses (PubMed:<a href="http://www.uniprot.org/citations/20434986" target="\_blank">20434986</a>, PubMed:<a href="http://www.uniprot.org/citations/20797629" target="\_blank">20797629</a>, PubMed:<a href="http://www.uniprot.org/citations/21138416" target="\_blank">21138416</a>, PubMed:<a href="http://www.uniprot.org/citations/30337470" target="\_blank">30337470</a>, PubMed:<a href="http://www.uniprot.org/citations/9346484" target="\_blank">9346484</a>). Acts as a part of the canonical IKK complex in the conventional pathway of NF-kappa-B activation (PubMed:<a href="http://www.uniprot.org/citations/9346484" target="\_blank">9346484</a>). Phosphorylates inhibitors of NF-kappa-B on 2 critical serine

residues (PubMed:<a href="http://www.uniprot.org/citations/20434986" target="\_blank">20434986</a>, PubMed:<a href="http://www.uniprot.org/citations/20797629" target="\_blank">20797629</a>, PubMed:<a href="http://www.uniprot.org/citations/21138416" target="\_blank">21138416</a>, PubMed:<a href="http://www.uniprot.org/citations/9346484" target="\_blank">9346484</a>). These modifications allow polyubiquitination of the inhibitors and subsequent degradation by the proteasome (PubMed:<a href="http://www.uniprot.org/citations/20434986" target="\_blank">20434986</a>, PubMed:<a href="http://www.uniprot.org/citations/20797629" target="\_blank">20797629</a>, PubMed:<a href="http://www.uniprot.org/citations/21138416" target="\_blank">21138416</a>, PubMed:<a href="http://www.uniprot.org/citations/9346484" target="\_blank">9346484</a>). In turn, free NF-kappa-B is translocated into the nucleus and activates the transcription of hundreds of genes involved in immune response, growth control, or protection against apoptosis (PubMed:<a href="http://www.uniprot.org/citations/20434986" target="\_blank">20434986</a>, PubMed:<a href="http://www.uniprot.org/citations/20797629" target="\_blank">20797629</a>, PubMed:<a href="http://www.uniprot.org/citations/21138416" target="\_blank">21138416</a>, PubMed:<a href="http://www.uniprot.org/citations/9346484" target="\_blank">9346484</a>). In addition to the NF-kappa-B inhibitors, phosphorylates several other components of the signaling pathway including NEMO/IKBKG, NF-kappa-B subunits RELA and NFKB1, as well as IKK-related kinases TBK1 and IKBKE (PubMed:<a href="http://www.uniprot.org/citations/11297557" target="\_blank">11297557</a>, PubMed:<a href="http://www.uniprot.org/citations/14673179" target="\_blank">14673179</a>, PubMed:<a href="http://www.uniprot.org/citations/20410276" target="\_blank">20410276</a>, PubMed:<a href="http://www.uniprot.org/citations/21138416" target="\_blank">21138416</a>). IKK-related kinase phosphorylations may prevent the overproduction of inflammatory mediators since they exert a negative regulation on canonical IKKs (PubMed:<a href="http://www.uniprot.org/citations/11297557" target="\_blank">11297557</a>, PubMed:<a href="http://www.uniprot.org/citations/20410276" target="\_blank">20410276</a>, PubMed:<a href="http://www.uniprot.org/citations/21138416" target="\_blank">21138416</a>). Phosphorylates FOXO3, mediating the TNF-dependent inactivation of this pro-apoptotic transcription factor (PubMed:<a href="http://www.uniprot.org/citations/15084260" target="\_blank">15084260</a>). Also phosphorylates other substrates including NAA10, NCOA3, BCL10 and IRS1 (PubMed:<a href="http://www.uniprot.org/citations/17213322" target="\_blank">17213322</a>, PubMed:<a href="http://www.uniprot.org/citations/19716809" target="\_blank">19716809</a>). Phosphorylates RIPK1 at 'Ser-25' which represses its kinase activity and consequently prevents TNF- mediated RIPK1-dependent cell death (By similarity). Phosphorylates the C-terminus of IRF5, stimulating IRF5 homodimerization and translocation into the nucleus (PubMed:<a href="http://www.uniprot.org/citations/25326418" target="\_blank">25326418</a>). Following bacterial lipopolysaccharide (LPS)-induced TLR4 endocytosis, phosphorylates STAT1 at 'Thr-749' which restricts interferon signaling and anti-inflammatory responses and promotes innate inflammatory responses (PubMed:<a href="http://www.uniprot.org/citations/38621137" target="\_blank">38621137</a>). IKBKB-mediated phosphorylation of STAT1 at 'Thr-749' promotes binding of STAT1 to the ARID5A promoter, resulting in transcriptional activation of ARID5A and subsequent ARID5A-mediated stabilization of IL6 (PubMed:<a href="http://www.uniprot.org/citations/32209697" target="\_blank">32209697</a>). It also promotes binding of STAT1 to the IL12B promoter and activation of IL12B transcription (PubMed:<a href="http://www.uniprot.org/citations/32209697" target="\_blank">32209697</a>).

### Cellular Location

Cytoplasm. Nucleus. Membrane raft. Note=Colocalized with DPP4 in membrane rafts.

### Tissue Location

Highly expressed in heart, placenta, skeletal muscle, kidney, pancreas, spleen, thymus, prostate, testis and peripheral blood

### Phospho-IKKb(Y609) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

### **Phospho-IKKB(Y609) Blocking Peptide - Images**

### **Phospho-IKKB(Y609) Blocking Peptide - Background**

NFKB1 (MIM 164011) or NFKB2 (MIM 164012) is bound to REL (MIM 164910), RELA (MIM 164014), or RELB (MIM 604758) to form the NFKB complex. The NFKB complex is 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 kinases (IKBKA, MIM 600664, or IKBKB) marks them for destruction via the ubiquitination pathway, thereby allowing activation of the NF-kappa-B 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).

### **Phospho-IKKB(Y609) Blocking Peptide - References**

- Yatherajam, G., et al. J. Immunol. 185(5):2665-2669(2010)  
Kenneth, N.S., et al. EMBO J. 29(17):2966-2978(2010)  
Zhao, M., et al. J. Biol. Chem. 285(32):24372-24380(2010)  
Niida, M., et al. Mol. Immunol. 47(14):2378-2387(2010)  
Schuurhof, A., et al. Pediatr. Pulmonol. 45(6):608-613(2010)