

**IKK alpha/IKK beta Antibody (Center) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP19253c****Specification**

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**IKK alpha/IKK beta Antibody (Center) Blocking peptide - Product Information**Primary Accession [O15111](#)**IKK alpha/IKK beta Antibody (Center) Blocking peptide - Additional Information**

Gene ID 1147

**Other Names**

Inhibitor of nuclear factor kappa-B kinase subunit alpha, I-kappa-B kinase alpha, IKK-A, IKK-alpha, IKBKA, IkappaB kinase, Conserved helix-loop-helix ubiquitous kinase, I-kappa-B kinase 1, IKK1, Nuclear factor NF-kappa-B inhibitor kinase alpha, NFKBIKA, Transcription factor 16, TCF-16, CHUK, IKKA, TCF16

**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.

**IKK alpha/IKK beta Antibody (Center) Blocking peptide - Protein Information**

Name CHUK

Synonyms IKKA, TCF16

**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: [18626576](http://www.uniprot.org/citations/18626576), PubMed: [9244310](http://www.uniprot.org/citations/9244310), PubMed: [9252186](http://www.uniprot.org/citations/9252186), PubMed: [9346484](http://www.uniprot.org/citations/9346484)). Acts as a part of the canonical IKK complex in the conventional pathway of NF-kappa-B activation and phosphorylates inhibitors of NF-kappa-B on serine residues (PubMed: [18626576](http://www.uniprot.org/citations/18626576), PubMed: [35952808](http://www.uniprot.org/citations/35952808), PubMed: [9244310](http://www.uniprot.org/citations/9244310), PubMed: [9252186](http://www.uniprot.org/citations/9252186), PubMed: [9346484](http://www.uniprot.org/citations/9346484)).

These modifications allow polyubiquitination of the inhibitors and subsequent degradation by the proteasome (PubMed:<a href="http://www.uniprot.org/citations/18626576" target="\_blank">18626576</a>, PubMed:<a href="http://www.uniprot.org/citations/9244310" target="\_blank">9244310</a>, PubMed:<a href="http://www.uniprot.org/citations/9252186" target="\_blank">9252186</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/18626576" target="\_blank">18626576</a>, PubMed:<a href="http://www.uniprot.org/citations/9244310" target="\_blank">9244310</a>, PubMed:<a href="http://www.uniprot.org/citations/9252186" target="\_blank">9252186</a>, PubMed:<a href="http://www.uniprot.org/citations/9346484" target="\_blank">9346484</a>). Negatively regulates the pathway by phosphorylating the scaffold protein TAXBP1 and thus promoting the assembly of the A20/TNFAIP3 ubiquitin-editing complex (composed of A20/TNFAIP3, TAX1BP1, and the E3 ligases ITCH and RNF11) (PubMed:<a href="http://www.uniprot.org/citations/21765415" target="\_blank">21765415</a>). Therefore, CHUK plays a key role in the negative feedback of NF-kappa-B canonical signaling to limit inflammatory gene activation. As part of the non-canonical pathway of NF-kappa-B activation, 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 (PubMed:<a href="http://www.uniprot.org/citations/20501937" target="\_blank">20501937</a>). In turn, these complexes regulate genes encoding molecules involved in B-cell survival and lymphoid organogenesis. Also participates in the negative feedback of the non-canonical NF-kappa-B signaling pathway by phosphorylating and destabilizing MAP3K14/NIK. Within the nucleus, phosphorylates CREBBP and consequently increases both its transcriptional and histone acetyltransferase activities (PubMed:<a href="http://www.uniprot.org/citations/17434128" target="\_blank">17434128</a>). Modulates chromatin accessibility at NF-kappa-B- responsive promoters by phosphorylating histones H3 at 'Ser-10' that are subsequently acetylated at 'Lys-14' by CREBBP (PubMed:<a href="http://www.uniprot.org/citations/12789342" target="\_blank">12789342</a>). Additionally, phosphorylates the CREBBP-interacting protein NCOA3. Also phosphorylates FOXO3 and may regulate this pro-apoptotic transcription factor (PubMed:<a href="http://www.uniprot.org/citations/15084260" target="\_blank">15084260</a>). Phosphorylates RIPK1 at 'Ser-25' which represses its kinase activity and consequently prevents TNF-mediated RIPK1-dependent cell death (By similarity). Phosphorylates AMBRA1 following mitophagy induction, promoting AMBRA1 interaction with ATG8 family proteins and its mitophagic activity (PubMed:<a href="http://www.uniprot.org/citations/30217973" target="\_blank">30217973</a>).

#### Cellular Location

Cytoplasm. Nucleus Note=Shuttles between the cytoplasm and the nucleus

#### Tissue Location

Widely expressed.

### IKK alpha/IKK beta Antibody (Center) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

### IKK alpha/IKK beta Antibody (Center) Blocking peptide - Images

### IKK alpha/IKK beta Antibody (Center) Blocking peptide - Background

This gene encodes a member of the serine/threonine protein kinase family. The encoded protein, a component of acytokine-activated protein complex that is an inhibitor of the essential transcription factor NF-kappa-B complex, phosphorylates sites that trigger the degradation of the inhibitor via

the ubiquitination pathway, thereby activating the transcription factor.

### **IKK alpha/IKK beta Antibody (Center) Blocking peptide - References**

Lahtela, J., et al. N. Engl. J. Med. 363(17):1631-1637(2010) Gouin, E., et al. Proc. Natl. Acad. Sci. U.S.A. 107(40):17333-17338(2010) Cummins, E.P., et al. J. Immunol. 185(7):4439-4445(2010) Li, T., et al. Nat. Immunol. 11(9):799-805(2010) Rotman, Y., et al. Hepatology 52(3):894-903(2010)