

**IKK beta Rabbit mAb**  
Catalog # AP75608**Specification****IKK beta Rabbit mAb - Product Information**

Application	<b>WB</b>
Primary Accession	<a href="#">O14920</a>
Reactivity	<b>Human</b>
Host	<b>Rabbit</b>
Clonality	<b>Monoclonal Antibody</b>
Calculated MW	<b>86564</b>

**IKK beta Rabbit mAb - Additional Information****Gene ID** 3551**Other Names**  
IKBKB**Dilution**  
WB~~1/500-1/1000**Format**  
Liquid**IKK beta Rabbit mAb - 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: [20434986](http://www.uniprot.org/citations/20434986), PubMed: [20797629](http://www.uniprot.org/citations/20797629), PubMed: [21138416](http://www.uniprot.org/citations/21138416), PubMed: [30337470](http://www.uniprot.org/citations/30337470), 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 (PubMed: [9346484](http://www.uniprot.org/citations/9346484)). Phosphorylates inhibitors of NF-kappa-B on 2 critical serine residues (PubMed: [20434986](http://www.uniprot.org/citations/20434986), PubMed: [20797629](http://www.uniprot.org/citations/20797629), PubMed: [21138416](http://www.uniprot.org/citations/21138416), PubMed: [9346484](http://www.uniprot.org/citations/9346484)). These modifications allow polyubiquitination of the inhibitors and subsequent degradation by the proteasome (PubMed: [20434986](http://www.uniprot.org/citations/20434986), PubMed: [20797629](http://www.uniprot.org/citations/20797629), PubMed: [21138416](http://www.uniprot.org/citations/21138416), PubMed: [9346484](http://www.uniprot.org/citations/9346484)).

<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 IKKBE (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>). IKKB-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

#### IKK beta Rabbit mAb - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)

- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

**IKK beta Rabbit mAb - Images**