

# IKK $\beta$ Polyclonal Antibody

Catalog # AP70486

## Product Information

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Application	WB, IHC-P
Primary Accession	<a href="#">O14920</a>
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	86564

## Additional Information

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Gene ID	3551
Other Names	IKBKB; IKKB; 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; NFKB1KB
Dilution	WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/5000. Not yet tested in other applications. IHC-P~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/5000. Not yet tested in other applications.
Format	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.
Storage Conditions	-20°C

## Protein Information

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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="#">20434986</a> , PubMed: <a href="#">20797629</a> , PubMed: <a href="#">21138416</a> , PubMed: <a href="#">30337470</a> , PubMed: <a href="#">9346484</a> ). Acts as a part of the canonical IKK complex in the conventional pathway of NF-kappa-B activation (PubMed: <a href="#">9346484</a> ). Phosphorylates inhibitors of NF-kappa-B on 2 critical serine residues (PubMed: <a href="#">20434986</a> , PubMed: <a href="#">20797629</a> , PubMed: <a href="#">21138416</a> , PubMed: <a href="#">9346484</a> ). These modifications allow polyubiquitination of the inhibitors and subsequent degradation by the proteasome (PubMed: <a href="#">20434986</a> , PubMed: <a href="#">20797629</a> , PubMed: <a href="#">21138416</a> , PubMed: <a href="#">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:[20434986](#), PubMed:[20797629](#), PubMed:[21138416](#), PubMed:[9346484](#)). 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:[11297557](#), PubMed:[14673179](#), PubMed:[20410276](#), PubMed:[21138416](#)). IKK-related kinase phosphorylations may prevent the overproduction of inflammatory mediators since they exert a negative regulation on canonical IKKs (PubMed:[11297557](#), PubMed:[20410276](#), PubMed:[21138416](#)). Phosphorylates FOXO3, mediating the TNF-dependent inactivation of this pro-apoptotic transcription factor (PubMed:[15084260](#)). Also phosphorylates other substrates including NAA10, NCOA3, BCL10 and IRS1 (PubMed:[17213322](#), PubMed:[19716809](#)). 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:[25326418](#)). 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:[38621137](#)). 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:[32209697](#)). It also promotes binding of STAT1 to the IL12B promoter and activation of IL12B transcription (PubMed:[32209697](#)).

#### Cellular Location

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

#### Tissue Location

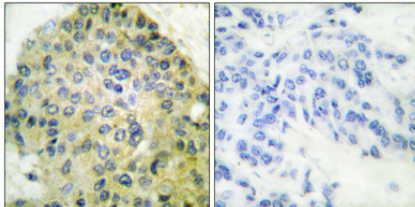
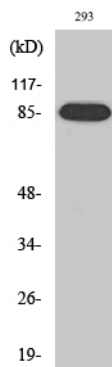
Highly expressed in heart, placenta, skeletal muscle, kidney, pancreas, spleen, thymus, prostate, testis and peripheral blood

## Background

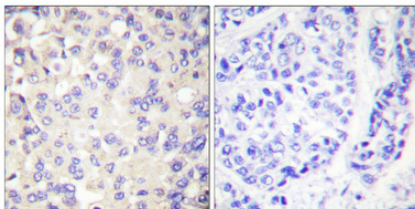
Serine kinase that plays an essential role in the NF- kappa-B signaling pathway which is activated by multiple stimuli such as inflammatory cytokines, bacterial or viral products, DNA damages or other cellular stresses (PubMed:[30337470](#)). Acts as part of the canonical IKK complex in the conventional pathway of NF-kappa-B activation. Phosphorylates inhibitors of NF-kappa-B on 2 critical serine residues. These modifications allow polyubiquitination of the inhibitors and subsequent degradation by the proteasome. 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. 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. IKK-related kinase phosphorylations may prevent the overproduction of inflammatory mediators since they exert a negative regulation on canonical IKKs. Phosphorylates FOXO3, mediating the TNF-dependent inactivation of this pro-apoptotic transcription factor. Also phosphorylates other substrates including NCOA3, BCL10 and IRS1. Within the nucleus, acts as an adapter protein for NFKBIA degradation in UV-induced NF-kappa-B activation.

## Images

Western Blot analysis of various cells using IKK $\beta$  Polyclonal Antibody



Immunohistochemical analysis of paraffin-embedded Human breast cancer. Antibody was diluted at 1:100(4°,overnight). High-pressure and temperature Tris-EDTA,pH8.0 was used for antigen retrieval. Negative contrl (right) obtaned from antibody was pre-absorbed by immunogen peptide.



Immunohistochemical analysis of paraffin-embedded Human breast cancer. Antibody was diluted at 1:100(4°,overnight). High-pressure and temperature Tris-EDTA,pH8.0 was used for antigen retrieval. Negative contrl (right) obtaned from antibody was pre-absorbed by immunogen peptide.

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