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Anti-IKKß (RABBIT) Antibody
IKK beta Antibody
Catalog # ASR3683

Specification

Anti-IKKß (RABBIT) Antibody - Product Information

Host	**Rabbit**
Conjugate	**Unconjugated**
Target Species	**Human**
Reactivity	**Rat, Human, Mouse**
Clonality	**Polyclonal**
Application	**WB, IHC, E, I, LCI**
Application Note	**This product was tested by immunoblot and found to be reactive against IKK-b at a dilution of 1:1000 followed by reaction with Peroxidase conjugated Affinity Purified anti-Rabbit IgG [H&L] (Goat) code #611-1302. Anti-IKKb is suitable for the detection by immunoblot of human, mouse and rat IKKb showing an 87 kDa band. Anti-IKKb has been tested in IHC using human placenta tissue.**
Physical State	**Liquid (sterile filtered)**
Immunogen	**IKKb peptide corresponding to the highly conserved C-terminus region of the human protein conjugated to Keyhole Limpet Hemocyanin (KLH).**
Preservative	**0.01% (w/v) Sodium Azide**

Anti-IKKß (RABBIT) Antibody - Additional Information

Gene ID 3551

Other Names
3551

Purity
This product was prepared from monospecific antiserum by delipidation and defibrination. Anti-IKKb may react non-specifically with other proteins. Control peptide (code #100-401-220p) will compete only with the specific reaction of antiserum with the IKKb subunit.

Storage Condition
Store vial at -20° C prior to opening. Aliquot contents and freeze at -20° C or below for extended storage. Avoid cycles of freezing and thawing. Centrifuge product if not completely clear after standing at room temperature. This product is stable for several weeks at 4° C as an undiluted liquid. Dilute only prior to immediate use.

Precautions Note
This product is for research use only and is not intended for therapeutic or diagnostic applications.

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Anti-IKKß (RABBIT) Antibody - 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, PubMed:20797629, PubMed:21138416, PubMed:30337470, PubMed:9346484). Acts as a part of the canonical IKK complex in the conventional pathway of NF-kappa-B activation (PubMed:9346484). Phosphorylates inhibitors of NF-kappa-B on 2 critical serine residues (PubMed:20434986, PubMed:20797629, PubMed:21138416, PubMed:9346484). These modifications allow polyubiquitination of the inhibitors and subsequent degradation by the proteasome (PubMed:20434986, PubMed:20797629, PubMed:21138416, PubMed:9346484). 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.

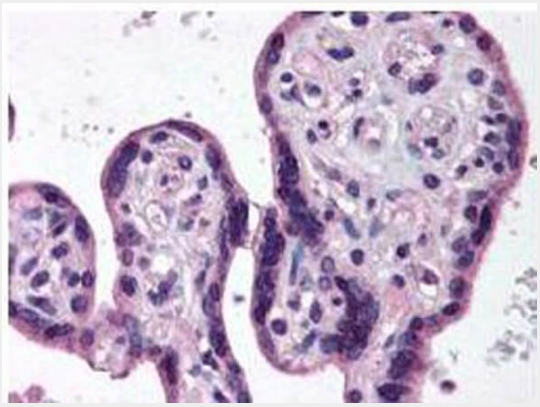
Tissue Location
Highly expressed in heart, placenta, skeletal muscle, kidney, pancreas, spleen, thymus, prostate, testis and peripheral blood

Anti-IKKß (RABBIT) Antibody - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Anti-IKKß (RABBIT) Antibody - Images



Immunohistochemistry of Anti-IKKß antibody. Tissue: human placenta was formalin fixed and paraffin embedded. No pre-treatment of sample was required. Primary Antibody: Anti-IKKß was diluted 1:500 to detect IKKß in tissue. The image shows the localization of antibody as the precipitated red signal, with a hematoxylin purple nuclear counter stain.

Anti-IKKß (RABBIT) Antibody - Background

NFkB comprises a family of cellular transcription factors that are involved in the inducible expression of a variety of cellular genes that regulate the inflammatory response and control of cell death. In the cytoplasm NFkB is negatively modulated by the inhibitory proteins IkB. In turn IkB is

phosphorylated by a cellular kinase complex called IKK. IKK is a heterodimer composed of two kinases: IKK-a and IKK-b that phosphorylate IkB leading to its degradation and the resulting translocation of NFkB to the nucleus. IKK kinase activity is modulated negatively by pharmaceutical agents such as aspirin and positively by various cellular components such as TNF- a, endotoxins and overexpression of cellular kinases like MEKK1. Aspirin appears to have its effect by inhibiting the binding of ATP to IKK.