

Anti-IKK α (RABBIT) Antibody
IKK alpha Antibody
Catalog # ASR3682**Specification**

Anti-IKK α (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	Anti-IKK α antibody was tested by immunoblot and found to be reactive against IKK alpha at a dilution of 1:1000 followed by reaction with Peroxidase conjugated Affinity Purified anti-Rabbit IgG [H&L] (Goat) code #611-1302. Anti-IKK α is suitable for the detection by immunoblot of human, mouse and rat IKK α showing an 85 kDa band. This product is tested in IHC. Liquid (sterile filtered)
Physical State	Liquid (sterile filtered)
Immunogen	IKK a 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-IKK α (RABBIT) Antibody - Additional Information**Gene ID** 1147**Other Names**
1147**Purity**

Anti-IKK α was prepared from monospecific antiserum by delipidation and defibrination. Anti- IKK a may react non-specifically with other proteins. Control peptide (code #100-401-219p) will compete only with the specific reaction of antiserum with the IKK a 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.

Anti-IKK α (RABBIT) Antibody - 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, PubMed:9244310, PubMed:9252186, PubMed: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, PubMed:35952808, PubMed:9244310, PubMed:9252186, PubMed:9346484). These modifications allow polyubiquitination of the inhibitors and subsequent degradation by the proteasome (PubMed:18626576, PubMed:9244310, PubMed:9252186, 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:18626576, PubMed:9244310, PubMed:9252186, PubMed:9346484). 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:21765415). 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:20501937). In turn, these complexes regulate genes encoding molecules involved in B-cell survival and lymphoid organogenesis. Participates also 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:17434128). 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:12789342). Additionally, phosphorylates the CREBBP-interacting protein NCOA3. Also phosphorylates FOXO3 and may regulate this pro- apoptotic transcription factor (PubMed:15084260). 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:30217973).

Cellular Location

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

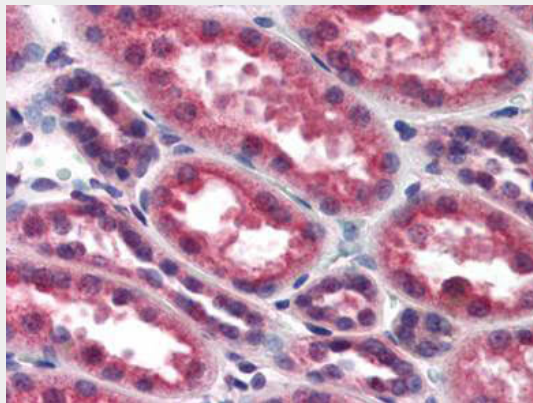
Tissue Location

Widely expressed.

Anti-IKK α (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 Cytometry](#)
- [Cell Culture](#)

Anti-IKK α (RABBIT) Antibody - Images

Rockland's Anti-IKK α antibody was diluted 1:500 to detect IKK α in human kidney tissue. Tissue was formalin fixed and paraffin embedded. No pre-treatment of sample was required. The image shows the localization of antibody as the precipitated red signal, with a hematoxylin purple nuclear counter stain.

Anti-IKK α (RABBIT) Antibody - Background

NF κ B 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 NF κ B is negatively modulated by the inhibitory proteins I κ B. In turn I κ B is phosphorylated by a cellular kinase complex called IKK. IKK is a heterodimer composed of two kinases: IKK-a and IKK-b that phosphorylate I κ B leading to its degradation and the resulting translocation of NF κ B to the nucleus. IKK kinase activity is modulated negatively by pharmaceutical agents such as aspirin and positively by various cellular components such as TNF- α , endotoxins and overexpression of cellular kinases like MEK1. Aspirin appears to have its effect by inhibiting the binding of ATP to IKK.