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# IKKα Polyclonal Antibody

Catalog # AP70483

## Product Information

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|---|---|
| **Application** | WB, IHC-P, IF, ICC, E |
| **Primary Accession** | O15111 |
| **Reactivity** | Human, Mouse, Rat |
| **Host** | Rabbit |
| **Clonality** | Polyclonal |
| **Calculated MW** | 84640 |

## Additional Information

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| **Gene ID** | 1147 |
| **Other Names** | CHUK; IKKA; TCF16; 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 |
| **Dilution** | WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/10000. Not yet tested in other applications. IHC-P~~N/A IF~~1:50~200 ICC~~N/A E~~N/A |
| **Format** | Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide. |
| **Storage Conditions** | -20℃ |

## Protein Information

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|---|---|
| **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. 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: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

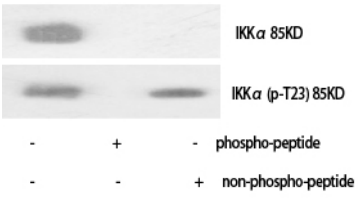
**Tissue Location** Widely expressed.

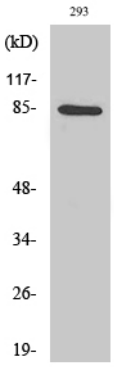
## Background

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. Acts as 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. 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. 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). 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. 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. 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. Additionally, phosphorylates the CREBBP-interacting protein NCOA3. Also phosphorylates FOXO3 and may regulate this pro-apoptotic transcription factor (PubMed:15084260).

## Images

Western Blot analysis of various cells using IKKα Polyclonal Antibody diluted at 1：1000




Western Blot analysis of 293 cells using IKKα Polyclonal Antibody diluted at 1：1000

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.