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[Marker Primary Antibody]

Rabbit Anti-IKK alpha/CHUK/AF647 Conjugated antibody

Catalog Number: SL2907R-AF647

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	Anti-IKK alpha/CHUK/AF647
Immunogen:	KLH conjugated synthetic peptide derived from human IKK alpha
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Applications:	Flow-Cyt=1ug/Test IF=1:100-500 ICC/IF=1:100-500 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human,Mouse,Rat(predicted:Dog,Horse)
Background:	background: Nuclear factor kappa B (NFkB) is a ubiquitous transcription factor and an essential mediator of gene expression during activation of immune and inflammatory responses. IκBα mediates the expression of a great variety of genes in response to extracellular stimuli including IL1, TNF alpha, and bacterial product LPS. NFkB is associated with IkB proteins in the cell cytoplasm, which inhibit IκBα activity. IKK is a serine protein kinase, and the IKK complex contains alpha and beta subunits (IKK alpha and IKK beta). IKK alpha and IKK beta interact with each other and both are essential for NFkB activation. IKK alpha specifically phosphorylates IkBa. IKKa is expressed in variety of human tissues. **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.

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	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. **Subunit:** Component of the I-kappa-B-kinase (IKK) core complex consisting of CHUK, IKBKB and IKBKG; probably four alpha/CHUK-beta/IKBKB dimers are associated with four gamma/IKBKG subunits. The IKK core complex seems to associate with regulatory or adapter proteins to form a IKK-signalosome holo-complex. The IKK complex associates with TERF2IP/RAP1, leading to promote IKK-mediated phosphorylation of RELA/p65. Part of a complex composed of NCOA2, NCOA3, CHUK/IKKA, IKBKB, IKBKG and CREBBP. Part of a 70-90 kDa complex at least consisting of CHUK/IKKA, IKBKB, NFKBIA, RELA, IKBKAP and MAP3K14. Directly interacts with IKK-gamma/NEMO and TRPC4AP (By similarity). May interact with TRAF2. Interacts with NALP2. May interact with MAVS/IPS1. Interacts with ARRB1 and ARRB2. Interacts with NLRC5; prevents CHUK phosphorylation and kinase activity. Interacts with PIAS1; this interaction

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	induces PIAS1 phosphorylation. **Subcellular Location:** Cytoplasm. Nucleus. Note=Shuttles between the cytoplasm and the nucleus. **Tissue Specificity:** Widely expressed. **Post-translational modifications:** Phosphorylated by MAP3K14/NIK, AKT and to a lesser extent by MEKK1, and dephosphorylated by PP2A. Autophosphorylated. Acetylation of Thr-179 by Yersinia yopJ prevents phosphorylation and activation, thus blocking the I-kappa-B signaling pathway. DISEASE: Defects in CHUK are the cause of cocoon syndrome (COCOS) [MIM:613630]; also known as fetal encasement syndrome. COCOS is a lethal syndrome characterized by multiple fetal malformations including defective face and seemingly absent limbs, which are bound to the trunk and encased under the skin. **Similarity:** Belongs to the protein kinase superfamily. Ser/Thr protein kinase family. I-kappa-B kinase subfamily. Contains 1 protein kinase domain. **Database links:** **Entrez Gene:** 1147 Human **Entrez Gene:** 309361 Rat Omim: 600664 Human **SwissProt:** O15111 Human **SwissProt:** Q60680 Mouse **Unigene:** 198998 Human **Unigene:** 3996 Mouse Important **Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.