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anti-IKK alpha antibody (AA 516-745)

2 Images



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Purification:

Quantity:	100 μL	
Target:	IKK alpha (CHUK)	
Binding Specificity:	AA 516-745	
Reactivity:	Human	
Host:	Rabbit	
Clonality:	Polyclonal	
Conjugate:	This IKK alpha antibody is un-conjugated	
Application:	Western Blotting (WB)	
Product Details		
Immunogen:	Recombinant fusion protein containing a sequence corresponding to amino acids 516-745 of human IKKalpha (NP_001269.3).	
Sequence:	EKAIHYAEVG VIGYLEDQIM SLHAEIMELQ KSPYGRRQGD LMESLEQRAI DLYKQLKHRP SDHSYSDSTE MVKIIVHTVQ SQDRVLKELF GHLSKLLGCK QKIIDLLPKV EVALSNIKEA DNTVMFMQGK RQKEIWHLLK IACTQSSARS LVGSSLEGAV TPQTSAWLPP TSAEHDHSLS CVVTPQDGET SAQMIEENLN CLGHLSTIIH EANEEQGNSM MNLDWSWLTE	
Isotype:	IgG	
Cross-Reactivity:	Human, Mouse, Rat	
Characteristics:	Polyclonal Antibodies	
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Affinity purification

Target Details

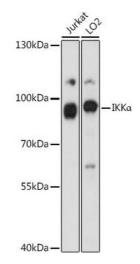
Target:	IKK alpha (CHUK)	
Alternative Name:	CHUK (CHUK Products)	
Background:	This gene encodes a member of the serine/threonine protein kinase family. The encoded	
	protein, a component of a cytokine-activated protein complex that is an inhibitor of the	
	essential transcription factor NF-kappa-B complex, phosphorylates sites that trigger the	
	degradation of the inhibitor via the ubiquination pathway, thereby activating the transcription	
	factor.,CHUK,IKBKA,IKK-alpha,IKK1,IKKA,NFKBIKA,TCF16,IKK alpha,Epigenetics & Nuclear	
	Signaling,Chromatin Modifying Enzymes,Phosphorylation,Signal	
	Transduction,Kinase,Serine/threonine kinases,PI3K-Akt Signaling Pathway,Cell Biology &	
	Developmental Biology, Apoptosis, Inhibition of Apoptosis, Death Receptor Signaling	
	Pathway,Endocrine & Metabolism,Insulin Receptor Signaling Pathway,Immunology &	
	Inflammation,B Cell Receptor Signaling Pathway,T Cell Receptor Signaling Pathway,NF-kB	
	Signaling Pathway, Toll-like Receptor Signaling Pathway, Cell Intrinsic Innate Immunity Signaling	
	Pathway,TLR Signaling,Cardiovascular,CHUK	
Molecular Weight:	84 kDa	
Gene ID:	1147	
UniProt:	015111	
Pathways:	PI3K-Akt Signaling, NF-kappaB Signaling, RTK Signaling, TCR Signaling, TLR Signaling, Fc-	
	epsilon Receptor Signaling Pathway, EGFR Signaling Pathway, Neurotrophin Signaling Pathway,	
	Activation of Innate immune Response, Hepatitis C, Toll-Like Receptors Cascades, BCR	
	Signaling, Ubiquitin Proteasome Pathway, S100 Proteins	

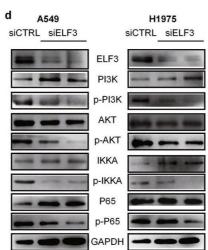
Application Details

Application Notes:	WB,1:500 - 1:2000	
Restrictions:	For Research Use only	
Handling		
Format:	Liquid	
Buffer:	PBS with 0.02 % sodium azide,50 % glycerol, pH 7.3.	
Preservative:	Sodium azide	
Precaution of Use:	This product contains Sodium azide: a POISONOUS AND HAZARDOUS SUBSTANCE which	

	should be handled by trained staff only.
Storage:	-20 °C
Storage Comment:	Store at -20°C. Avoid freeze / thaw cycles

Images





Western Blotting

Image 1. Western blot analysis of extracts of various cell lines, using IKKα antibody (ABIN6132067, ABIN6138615, ABIN6138616 and ABIN6213841) at 1:1000 dilution. Secondary antibody: HRP Goat Anti-Rabbit IgG (H+L) (ABIN1684268 and ABIN3020597) at 1:10000 dilution. Lysates/proteins: 25 μg per lane. Blocking buffer: 3 % nonfat dry milk in TBST. Detection: ECL Basic Kit (RM00020). Exposure time: 30s.

Western Blotting

Image 2. ELF3 can be induced by IL1B and promotes tumor growth through PI3K/AKT/NF-kB pathway.a Representative immunostaining showed that ELF3 expression in LUAD6 and LUAD7 is higher than that in the matched normal lung tissues. b Expression of ELF3 and selected NF-kB target genes in additional 12 LUAD tumor samples by gRT-PCR. The plot showed that the median expression of ELF3 and NF-KB targeting genes related to proliferation and antiapoptosis are higher in the tumor tissues than their matched normal lung tissues. Colored dots refer to different individuals. c Expression of ELF3, NFKB1, and selected NF-к B target genes in NCI-H1975 (top) and A549 (bottom) cell lines by gRT-PCR after knockdown of ELF3 by siRNA (The controls were transfected with control siRNA (siCTRL)). In both cell lines, downregulation of ELF3 results in decreased expression of BCL2L1, CCND1, and PTGS2 which are responsible for anti-apoptosis, proliferation, and inflammation in tumor cells respectively. d Western blot

showing the protein and phosphorylation levels of the key components in PI3K/AKT/NF-кВ pathway in NCI-H1975 and A549 cells transfected with siELF3 or siCTRL. The phosphorylation levels of PI3K, AKT, IKKa, and P65 were decreased after ELF3 knockdown with unaltered total protein levels. e qRT-PCR showing the expression levels of ELF3, NFKB1, and selected NF-kB target genes in NCI-H1975 (top) and A549 cells (bottom) treated with 10 ng/mL IL1B for 1h. The plots showed that the expression of ELF3 and NF-kB target genes such as BCL2L1, CCND1, PTGS2, and ICAM1 are increased in both cell lines after IL1B treatment. f qRT-PCR showing expression of ELF3, NFKB1, and the NF-kB target genes in NCI-H1975 (top) and A549 cells (bottom) which were transfected with siELF3 or siCTRL before being treated with 10 ng/mL IL1B for 1h. The plots showed that IL1B induced upregulation of NF-KB target genes are compromised by ELF3 knockdown in both cell lines. - figure provided by CiteAb. Source: PMID33144684