



# IκB-α Polyclonal Antibody

<b>Catalog No</b>	YP-Ab-01830
<b>Isotype</b>	IgG
<b>Reactivity</b>	Human;Mouse;Rat
<b>Applications</b>	WB;IHC;IF;ELISA
<b>Gene Name</b>	NFKBIA IKBA MAD3 NFKBI
<b>Protein Name</b>	NF-kappa-B inhibitor alpha
<b>Immunogen</b>	The antiserum was produced against synthesized peptide derived from human IκappaB-alpha. AA range:15-64
<b>Specificity</b>	IκB-α Polyclonal Antibody detects endogenous levels of IκB-α protein.
<b>Formulation</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
<b>Source</b>	Polyclonal, Rabbit,IgG
<b>Purification</b>	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
<b>Dilution</b>	Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/10000. Not yet tested in other applications.
<b>Concentration</b>	1 mg/ml
<b>Purity</b>	≥90%
<b>Storage Stability</b>	-20°C/1 year
<b>Synonyms</b>	NFKBIA; IKBA; MAD3; NFKBI; NF-kappa-B inhibitor alpha; I-kappa-B-alpha; IκB-alpha; IκappaBalpha; Major histocompatibility complex enhancer-binding protein MAD3
<b>Observed Band</b>	about 40kd
<b>Cell Pathway</b>	Cytoplasm. Nucleus. Shuttles between the nucleus and the cytoplasm by a nuclear localization signal (NLS) and a CRM1-dependent nuclear export. .
<b>Tissue Specificity</b>	Brain,Kidney,Lymph node,Monocyte,
<b>Function</b>	disease:Defects in NFKBIA are the cause of ectodermal dysplasia anhidrotic with T-cell immunodeficiency autosomal dominant (AEDAID) [MIM:612132]. Ectodermal dysplasia defines a heterogeneous group of disorders due to abnormal development of two or more ectodermal structures. AEDAID is an ectodermal dysplasia associated with decreased production of pro-inflammatory cytokines and certain interferons, rendering patients susceptible to infection.,function:Inhibits the activity of dimeric NF-kappa-B/REL complexes by trapping REL dimers in the cytoplasm through masking of their nuclear localization signals. On cellular stimulation by immune and proinflammatory responses, becomes phosphorylated promoting ubiquitination and degradation, enabling the dimeric RELA to translocate to the nucleus and activate



transcription.,induction:Induced in adherent monocytes.,online  
information:NFKBIA mutation

**Background**

This gene encodes a member of the NF-kappa-B inhibitor family, which contain multiple ankrin repeat domains. The encoded protein interacts with REL dimers to inhibit NF-kappa-B/REL complexes which are involved in inflammatory responses. The encoded protein moves between the cytoplasm and the nucleus via a nuclear localization signal and CRM1-mediated nuclear export. Mutations in this gene have been found in ectodermal dysplasia anhidrotic with T-cell immunodeficiency autosomal dominant disease. [provided by RefSeq, Aug 2011],

**matters needing attention**

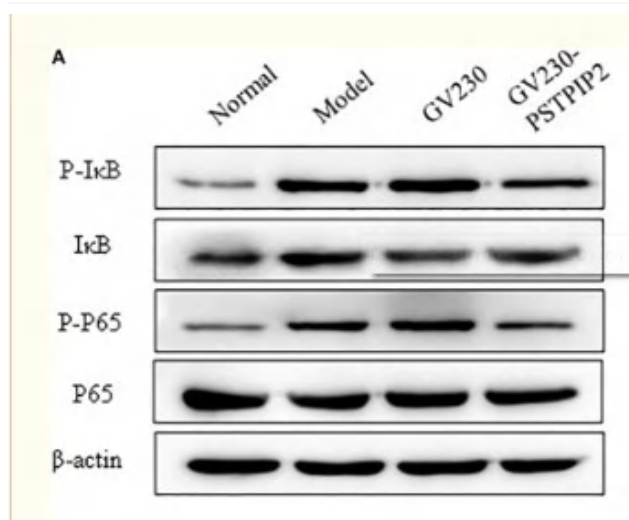
Avoid repeated freezing and thawing!

**Usage suggestions**

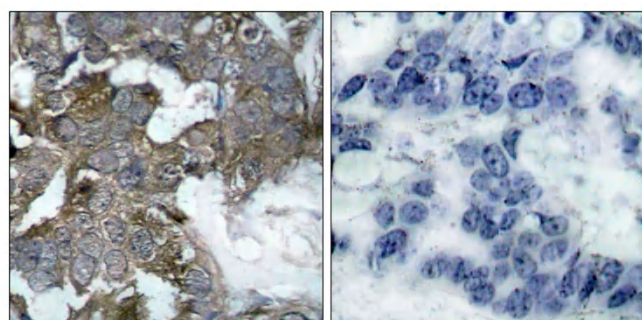
This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.



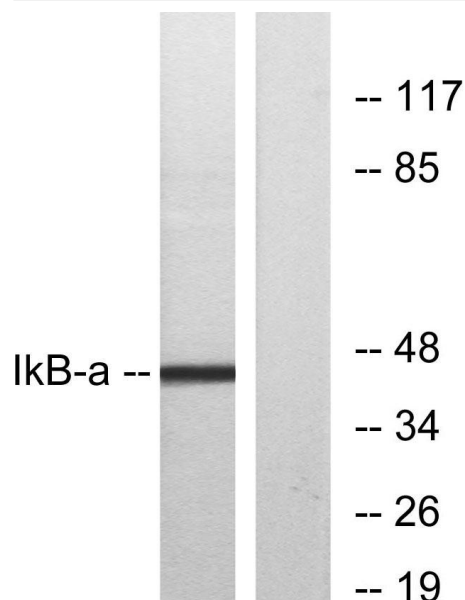
## Products Images



Yao, Yao, et al. "PSTPIP2 inhibits the inflammatory response and proliferation of fibroblast-like synoviocytes in vitro." *Frontiers in pharmacology* 9 (2018): 1432.



Immunohistochemistry analysis of paraffin-embedded human breast carcinoma tissue, using IκB-α Antibody. The picture on the right is blocked with the synthesized peptide.



Western blot analysis of lysates from MCF7 cells, treated with TNF-α, using IκB-α Antibody. The lane on the right is blocked with the synthesized peptide.