

IκB-α (Phospho Tyr42) Rabbit pAb

CatalogNo: YP0152

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse, Rat

Applications

- WB, IHC, IF, ELISA

MW

- about 40kD (Observed)

Isotype

- IgG

Recommended Dilution Ratios

WB 1:500-1:2000

IHC 1:100-1:300

ELISA 1:10000

IF 1:50-200

Storage

Storage*

-15°C to -25°C/1 year (Do not lower than -25°C)

Formulation

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

Basic Information

Clonality

Polyclonal

Immunogen Information

Immunogen

The antiserum was produced against synthesized peptide derived from human IκappaB-alpha around the phosphorylation site of Tyr42. AA range:9-58

Specificity Phospho-IkB-α (Y42) Polyclonal Antibody detects endogenous levels of IkB-α protein only when phosphorylated at Y42. The name of modified sites may be influenced by many factors, such as species (the modified site was not originally found in human samples) and the change of protein sequence (the previous protein sequence is incomplete, and the protein sequence may be prolonged with the development of protein sequencing technology). When naming, we will use the "numbers" in historical reference to keep the sites consistent with the reports. The antibody binds to the following modification sequence (lowercase letters are modification sites):EEyEQ

| Target Information

Gene name NFKBIA IKBA MAD3 NFKBI

Protein Name NF-kappa-B inhibitor alpha

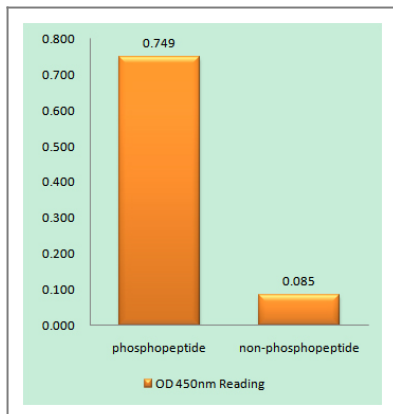
Organism	Gene ID	UniProt ID
Human	4792;	P25963;
Mouse	18035;	Q9Z1E3;
Rat	25493;	Q63746;

Cellular Localization Cytoplasm. Nucleus. Shuttles between the nucleus and the cytoplasm by a nuclear localization signal (NLS) and a CRM1-dependent nuclear export. .

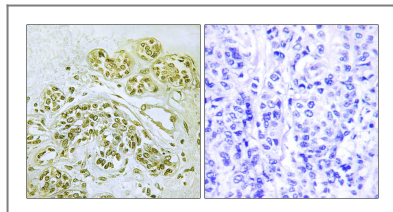
Tissue specificity Brain,Kidney,Lymph node,Monocyte,

Function Disease:Defects in NFKBIA are the cause of ectodermal dysplasia anhidrotic with T-cell immunodeficiency autosomal dominant (AEDDAID) [MIM:612132]. Ectodermal dysplasia defines a heterogeneous group of disorders due to abnormal development of two or more ectodermal structures. AEDDAID 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 tranlocate to the nucleus and activate transcription.,induction:Induced in adherent monocytes.,online information:NFKBIA mutation db,PTM:Phosphorylated; disables inhibition of NF-kappa-B DNA-binding activity.,PTM:Sumoylated; sumoylation requires the presence of the nuclear import signal.,PTM:Ubiquitinated; subsequent to stimulus-dependent phosphorylation on serines.,similarity:Belongs to the NF-kappa-B inhibitor family.,similarity:Contains 5 ANK repeats.,subcellular location:Shuttles between the nucleus and the cytoplasm by a nuclear localization signal (NLS) and a CRM1-dependent nuclear export.,subunit:Interacts with RELA; the interaction requires the nuclear import signal. Interacts with NKIRAS1 and NKIRAS2. Part of a 70-90 kDa complex at least consisting of CHUK, IKBKB, NFKBIA, RELA, IKBKAP and MAP3K14. Interacts with HBV protein X. Interacts with RWDD3; the interaction enhances sumoylation.,

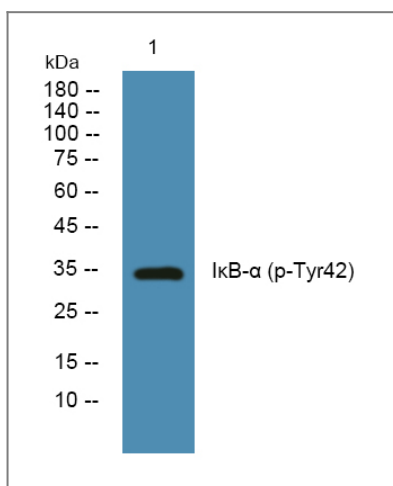
| Validation Data



Enzyme-Linked Immunosorbent Assay (Phospho-ELISA) for Immunogen Phosphopeptide (Phospho-left) and Non-Phosphopeptide (Phospho-right), using IkappaB-alpha (Phospho-Tyr42) Antibody



Immunohistochemistry analysis of paraffin-embedded human breast carcinoma, using IkappaB-alpha (Phospho-Tyr42) Antibody. The picture on the right is blocked with the phospho peptide.



Western blot analysis of lysates from Jurkat cells, primary antibody was diluted at 1:1000, 4° over night

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