

IκB-α (Phospho Ser32) (PT0899R) PT™ Rabbit mAb

CatalogNo: YM8774 **Recombinant** 

Key Features

Host Species

- Rabbit

Reactivity

- Human, Mouse, Rat

Applications

- WB, IF, IP, ELISA

MW

- 36kD (Calculated)
40kD (Observed)

Isotype

- IgG, Kappa

Storage

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

Formulation PBS, 50% glycerol, 0.05% Proclin 300, 0.05% BSA

Recommended Dilution Ratios

WB 1:2000-1:10000

IF 1:200-1:1000

ELISA 1:5000-1:20000

IP 1:50-1:200

Basic Information

Clonality Monoclonal

Clone Number PT0899R

Immunogen Information

Specificity

IκB-α (Phospho Ser32) antibody detects endogenous levels of IκB-α only when phosphorylated at Ser32 and dually phosphorylated at two sites. 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 When naming, we will use the "numbers" in historical reference to keep the sites consistent with the reports.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): HDsGL

| Target Information

Gene name NFKBIA IKBA MAD3 NFKBI

Protein Name NF-kappa-B inhibitor alpha

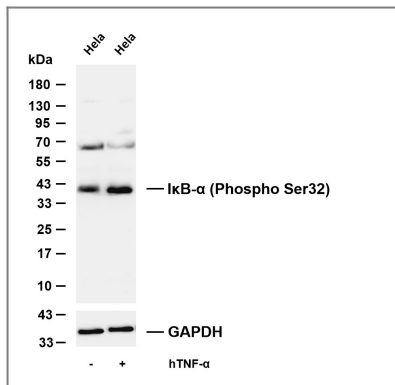
Organism	Gene ID	UniProt ID
Human	4792;	P25963;
Mouse	18035;	Q9Z1E3;
Rat	25493;	Q63746;

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

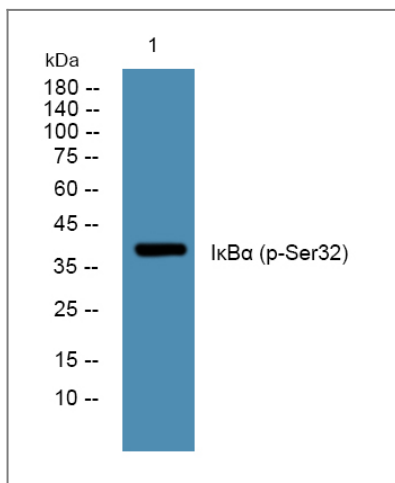
Tissue specificity Brain,Kidney,Lymph node,Monocyte,

Function Disease:Defects in NFKBIA are the cause of ectodermal dysplasia anhidrotic with T-cell immunodeficiency autosomal dominant (ADEDAID) [MIM:612132]. Ectodermal dysplasia defines a heterogeneous group of disorders due to abnormal development of two or more ectodermal structures. ADEDAID 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 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



Various whole cell lysates were separated by 4-8% SDS-PAGE, and the membrane was blotted with anti-protein name (PT0067R) antibody. The HRP-conjugated Goat anti-Rabbit IgG (H + L) antibody was used to detect the antibody. Lane 1: HeLa Lane 2: HeLa Predicted band size: 36kDa Observed band size: 40kDa



Western blot analysis of lysates from A431 cells, primary antibody was diluted at 1:1000, 4° over night

Contact information

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PT™ Rabbit mAb

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