

Cat. No:	ABN04890
Conjugate:	Unconjugated
Size:	100μL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human IκappaB-alpha around the phosphorylation site of Tyr305. AA range:268-317
Reactivity:	Human,Mouse,Rat,Monkey
Applications:	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:50-1:200,ELISA 1:5000-1:10000
Molecular Weight:	about 40kDa
Purification:	Affinity purification
Synonyms:	NFKBIA; IKBA; MAD3; NFKBI; NF-kappa-B inhibitor alpha; I-kappa-B-alpha; IκB-alpha; IκappaBalpha; Major histocompatibility complex enhancer-binding protein MAD3
Background:	<p>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],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 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.,</p>
Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.

Cat. No:	ABN04890
Conjugate:	Unconjugated
Size:	100μL
Clone:	Polyclonal
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human IκappaB-alpha around the phosphorylation site of Tyr305. AA range:268-317
Reactivity:	Human,Mouse,Rat,Monkey
Applications:	WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:50-1:200,ELISA 1:5000-1:10000
Molecular Weight:	about 40kDa
Purification:	Affinity purification
Synonyms:	NFKBIA; IKBA; MAD3; NFKBI; NF-kappa-B inhibitor alpha; I-kappa-B-alpha; IκB-alpha; IκappaBalpha; Major histocompatibility complex enhancer-binding protein MAD3

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],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 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.,

Form:	Liquid
Buffer:	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.

Storage: Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles.

**For Research use only
IMMUNOLOGICAL SCIENCES**