



IKK- γ Rabbit mAb

Catalog No	YP-rAb-17001
Isotype	IgG
Reactivity	Human,Mouse,Rat
Applications	WB,IHC,IF,IP,ELISA
Gene Name	IKBKG
Protein Name	NF-kappa-B essential modulator
Purification Process	Protein A
Specificity	Endogenous
Formulation	PBS, 50% glycerol, 0.05% Proclin 300, 0.05%BSA
Source	Monoclonal, Rabbit,IgG
Dilution	IHC 1:500-1:2000; WB 1:2000-1:10000; IF 1:200-1:1000; ELISA 1:5000-1:20000; IP 1:50-1:200; Note: For IHC, we suggest antigen retrieval with TE buffer pH 9.0
Concentration	0.5 mg/ml
Purity	≥90%
Storage Stability	-15° C to -25° C/1 year(Do not lower than -25° C)
Synonyms	IKBKG ; FIP3 ; NEMO ; NF-kappa-B essential modulator ; NEMO ; FIP-3 ; IκB kinase-associated protein 1 ; IKKAP1 ; Inhibitor of nuclear factor kappa-B kinase subunit gamma ; I-kappa-B kinase subunit gamma ; IKK-gamma ; IKKG ; IκB kinase subunit gamma ; NF
Observed Band	43-60kD
Calculated Molecular Weight	48kD
Cell Pathway	Cytoplasm . Nucleus . Sumoylated NEMO accumulates in the nucleus in response to genotoxic stress. .
Tissue Specificity	Heart, brain, placenta, lung, liver, skeletal muscle, kidney and pancreas.
Function	Caution:The sequence shown here is derived from an Ensembl automatic analysis pipeline and should be considered as preliminary data.,Disease:Defects in IKBKG are a cause of immunodeficiency without anhidrotic ectodermal dysplasia [MIM:300584]; also called isolated immunodeficiency or pure immunodeficiency. Patients manifest immunodeficiency not associated with other abnormalities, and resulting in increased infection susceptibility. Patients suffer from multiple episodes of infectious diseases.,Disease:Defects in IKBKG are the cause of ectodermal dysplasia anhidrotic with immunodeficiency X-linked (EDAXID) [MIM:300291]; also known as hypohidrotic ectodermal dysplasia with





immunodeficiency (HED-ID). Ectodermal dysplasia defines a heterogeneous group of disorders due to abnormal development of two or more ectodermal structures. EDAXID is characterized by absence of sweat glands, sparse scalp hair, rare conical teeth and immunological abnormalities resulting in severe infectious diseases. Disease: Defects in IKBKG are the cause of ectodermal dysplasia anhidrotic with immunodeficiency-osteopetrosis-lymphedema (OLEDAID) [MIM:300301]. Disease: Defects in IKBKG are the cause of incontinentia pigmenti (IP) [MIM:308300]; formerly designed familial incontinentia pigmenti type II (IP2). IP is a genodermatosis usually prenatally lethal in males. In affected females, it causes abnormalities of the skin, hair, eyes, nails, teeth, skeleton, heart, and central nervous system. The prominent skin signs occur in four classic cutaneous stages: perinatal inflammatory vesicles, verrucous patches, a distinctive pattern of hyperpigmentation and dermal scarring. Disease: Defects in IKBKG are the cause of recurrent isolated invasive pneumococcal disease type 2 (IPD2) [MIM:300640]. Recurrent invasive pneumococcal disease (IPD) is defined as two episodes of IPD occurring at least 1 month apart, whether caused by the same or different serotypes or strains. Recurrent IPD occurs in at least 2% of patients in most series, making IPD the most important known risk factor for subsequent IPD. Disease: Defects in IKBKG are the cause of susceptibility to X-linked familial atypical mycobacteriosis type 1 (AMCBX1) [MIM:300636]; also known as X-linked disseminated atypical mycobacterial infection type 1 or X-linked susceptibility to mycobacterial disease type 1. AMCBX1 is the X-linked recessive form of mendelian susceptibility to mycobacterial disease (MSMD). MSMD is a congenital syndrome resulting in predisposition to clinical disease caused by weakly virulent mycobacterial species, such as bacillus Calmette-Guerin vaccines and non-tuberculous, environmental mycobacteria. Patients are also susceptible to the more virulent species Mycobacterium tuberculosis. Function: Regulatory subunit of the IKK core complex which phosphorylates inhibitors of NF-kappa-B thus leading to the dissociation of the inhibitor/NF-kappa-B complex and ultimately the degradation of the inhibitor. Also considered to be a mediator for TAX activation of NF-kappa-B. Could be implicated in NF-kappa-B-mediated protection from cytokine toxicity. online information: IKBKG mutation db, PTM: Mono-ubiquitinated on Lys-277 and Lys-309; promotes nuclear export. PTM: Phosphorylation at Ser-68 attenuates aminoterminal homodimerization. PTM: Polyubiquitinated on Lys-285 through 'Lys-63'; the ubiquitination is mediated by NOD2 and RIPK2 and probably plays a role in signaling by facilitating interactions with ubiquitin domain-containing proteins and activates the NF-kappa-B pathway. PTM: Polyubiquitinated on Lys-399 through 'Lys-63'; the ubiquitination is mediated by BCL10, MALT1 and TRAF6 and probably plays a role in signaling by facilitating interactions with ubiquitin domain-containing proteins and activates the NF-kappa-B pathway. PTM: Sumoylated on Lys-277 and Lys-309 by SUMO1; the modification results in phosphorylation of Ser-85 by ATM leading to a replacement of the sumoylation by mono-ubiquitination on these residues. similarity: Contains 1 C2HC-type zinc finger. subunit: Homodimer; disulfide-linked. Component of the I-kappa-B-kinase (IKK) core complex consisting of CHUK, IKBKB and IKBKG; probably four alpha/CHUK-beta/IKBKB dimers are associated with four gamma/IKBKG subunits. The IKK core complex seems to associate with regulatory or adapter proteins to form a IKK-signalosome holo-complex. Part of a complex composed of NCOA2, NCOA3, CHUK/IKKA, IKBKB, IKBKG and CREBBP. Interacts with COPS3, CYLD, NALP2, TRPC4AP and LRDD. Interacts with ATM; the complex is exported from the nucleus. Interacts with TRAF6. Interacts with HTLV-1 Tax oncoprotein; the interaction activates IKBKG. Interacts with TANK; the interaction is enhanced by IKBKE and TBK1. Part of a ternary complex consisting of TANK, IKBKB and IKBKG. tissue specificity: Heart, brain, placenta, lung, liver, skeletal muscle, kidney and pancreas.

Background

This gene encodes the regulatory subunit of the inhibitor of kappaB kinase (IKK) complex, which activates NF-kappaB resulting in activation of genes involved in inflammation, immunity, cell survival, and other pathways. Mutations in this gene result in incontinentia pigmenti, hypohidrotic ectodermal dysplasia, and several other types of immunodeficiencies. A pseudogene highly similar to this locus is located in an adjacent region of the X chromosome. [provided by RefSeq, Mar 2016],

matters needing attention

Avoid repeated freezing and thawing!

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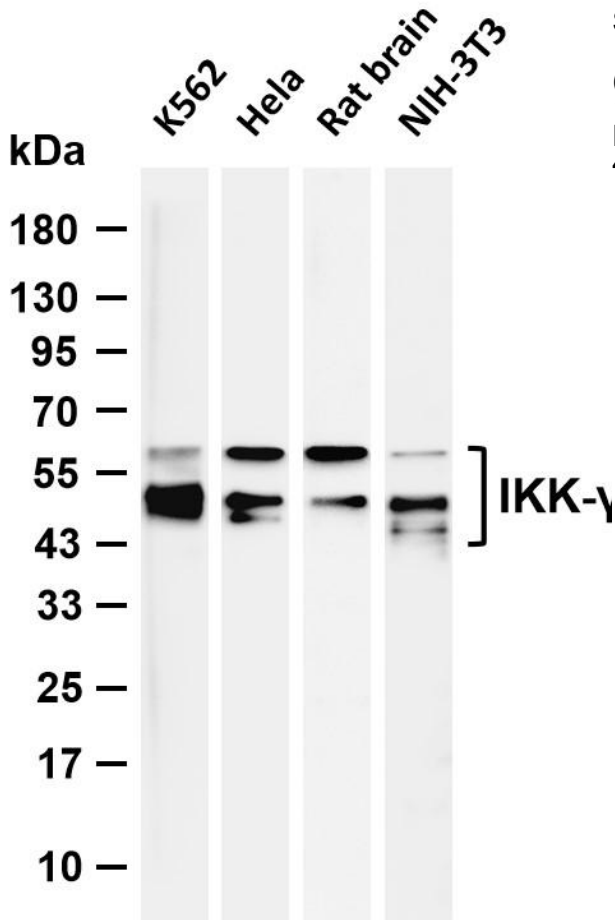


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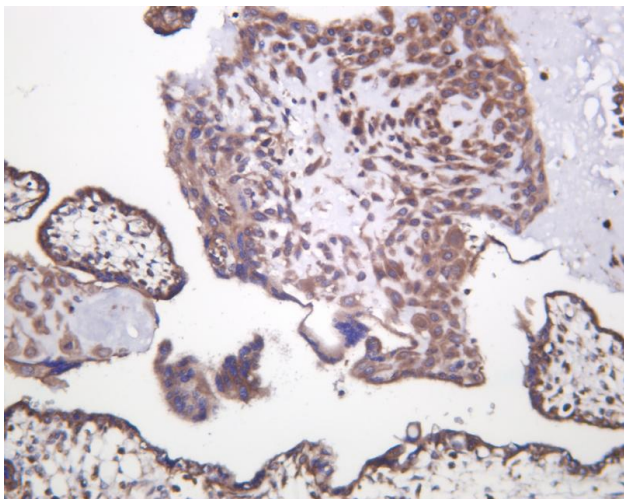


Usage suggestions

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



Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-IKK- γ antibody. The HRP-conjugated Goat anti-Rabbit IgG (H + L) antibody was used to detect the antibody. Lane 1: K562 Lane 2: HeLa Lane 3: Rat brain Lane 4: NIH-3T3
Predicted band size: 48kDa Observed band size: 43-60kDa



Human placenta was stained with anti-IKK- γ Rabbit antibody

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