

Product datasheet

Alexa Fluor® 647 Anti-IKK gamma/NEMO antibody [EPR16629] ab205788

Recombinant RabMAb

2 Images

exa Fluor® 647 Anti-IKK gamma/NEMO antibody [EPR16629] exa Fluor® 647 Rabbit monoclonal [EPR16629] to IKK gamma/NEMO abbit exa Fluor® 647. Ex: 652nm, Em: 668nm uitable for: ICC/IF eacts with: Human redicted to work with: Mouse, Rat
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ecombinant full length protein corresponding to Human IKK gamma/NEMO aa 1 to the C- rminus. atabase link: Q9Y6K9
C/IF: HeLa cells
is product is a recombinant monoclonal antibody, which offers several advantages including:
High batch-to-batch consistency and reproducibility mproved sensitivity and specificity .ong-term security of supply Animal-free production or more information <u>see here</u> . ur RabMAb [®] technology is a patented hybridoma-based technology for making rabbit
onoclonal antibodies. For details on our patents, please refer to <u>RabMAb[®] patents</u> .
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Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C. Avoid freeze / thaw cycle. Store In the Dark.
Storage buffer	pH: 7.40 Preservative: 0.02% Sodium azide Constituents: 1% BSA, 30% Glycerol (glycerin, glycerine), PBS
Purity	Protein A purified
Clonality	Monoclonal
Clone number	EPR16629
lsotype	lgG

Applications

The Abpromise guarantee Our <u>Abpromise guarantee</u> covers the use of ab205788 in the following tested applications.

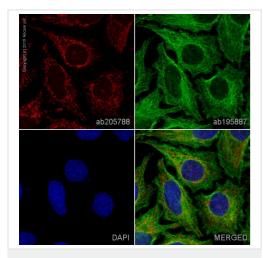
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
ICC/IF		1/100. This product gave a positive signal in HeLa cells fixed with 4% formaldehyde (10 min) and 100% methanol (5 min)

Target	
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 (By similarity). Essential for viral activation of IRF3.
Tissue specificity	Heart, brain, placenta, lung, liver, skeletal muscle, kidney and pancreas.
Involvement in disease	Defects in IKBKG are the cause of ectodermal dysplasia anhidrotic with immunodeficiency X- linked (EDAID) [MIM:300291]; also known as hypohidrotic ectodermal dysplasia with immunodeficiency (HED-ID). Is a form of ectoderma dysplasia, a heterogeneous group of disorders due to abnormal development of two or more ectodermal structures. Characterized by absence of sweat glands, sparse scalp hair, rare conical teeth and immunological abnormalities resulting in severe infectious diseases. Defects in IKBKG are the cause of ectodermal dysplasia anhidrotic with immunodeficiency- osteopetrosis-lymphedema (OLEDAID) [MIM:300301]. Defects in IKBKG are a cause of immunodeficiency NEMO-related without anhidrotic ectodermal

	dysplasia (NEMOID) [MIM:300584]; also called immunodeficiency without anhidrotic ectodermal dysplasia, 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. Defects in IKBKG are the cause of susceptibility to X-linked familial atypical micobacteriosis 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. 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. Defects in IKBKG are the cause of incontinentia pigmenti (IP) [MIM:308300]; formerly designed familial incontinentia pigment 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, verucous patches, a distinctive pattern of
Sequence similarities	hyperpigmentation and dermal scarring. Contains 1 C2HC-type zinc finger.
Domain	The leucine-zipper domain and the C2HC-type zinc-finger are essential for polyubiquitin binding and for the activation of IRF3.
Post-translational modifications	 Phosphorylation at Ser-68 attenuates aminoterminal homodimerization. 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. 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. Monoubiquitinated on Lys-277 and Lys-309; promotes nuclear export. Linear polyubiquitinated on Lys-285; the head-to-tail polyubiquitination is mediated by the LUBAC complex. Linear polyubiquitinated on Lys-309; the head-to-tail polyubiquitination is mediated by the LUBAC complex. 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.
Cellular localization	Cytoplasm. Nucleus. Sumoylated NEMO accumulates in the nucleus in response to genotoxic stress.

Images



Immunocytochemistry/ Immunofluorescence - Alexa Fluor® 647 Anti-IKK gamma/NEMO antibody [EPR16629] (ab205788) ab205788 staining IKK gamma/NEMO in HeLa cells. The cells were fixed with 100% methanol (5min), permeabilized with 0.1% Triton X-100 for 5 minutes and then blocked with 1% BSA/10% normal goat serum/0.3M glycine in 0.1% PBS-Tween for 1h. The cells were then incubated overnight at +4°C with ab205788 at 1/100 dilution (shown in red) and **ab195887**, Mouse monoclonal to alpha Tubulin (Alexa Fluor[®] 488), at 1/250 dilution (shown in green). Nuclear DNA was labelled with DAPI (shown in blue).

Image was taken with a confocal microscope (Leica-Microsystems, TCS SP8).

This product also gave a positive signal under the same testing conditions in HeLa cells fixed with 4% formaldehyde (10 min).



[EPR16629] (ab205788)

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