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Anti-Rel B Antibody

Catalog # ABO11111

Product Information

Application	WB
Primary Accession	Q01201
Host	Rabbit
Reactivity	Human, Mouse, Rat
Clonality	Polyclonal
Format	Lyophilized
Description	Rabbit IgG polyclonal antibody for Transcription factor RelB(RELB) detection. Tested with WB in Human;Mouse;Rat.
Reconstitution	Add 0.2ml of distilled water will yield a concentration of 500ug/ml.

Additional Information

Gene ID	5971
Other Names	Transcription factor RelB, I-Rel, RELB
Calculated MW	62134
Application Details	Western blot, 0.1-0.5 □g/ml, Human, Mouse, Rat
Subcellular Localization	Nucleus . Cytoplasm, cytoskeleton, microtubule organizing center, centrosome . Colocalizes with NEK6 in the centrosome.
Source	Eukaryota
Protein Name	Transcription factor RelB
Contents	Each vial contains 5mg BSA, 0.9mg NaCl, 0.2mg Na2HPO4, 0.05mg Thimerosal, 0.05mg NaN3.
Immunogen	A synthetic peptide corresponding to a sequence at the C-terminus of human Rel B(393-413aa LPFTYLPRDHDSYGVDKKRKR), identical to the related mouse sequence.
Purification	Immunogen affinity purified.
Cross Reactivity	No cross reactivity with other proteins
Storage	At -20˚C for one year. After r˚Constitution, at 4˚C for one month. It˚Can also be aliquotted and stored frozen at -20˚C for a longer time.Avoid repeated freezing and thawing.
Sequence Similarities	Contains 1 RHD (Rel-like) domain.

Protein Information

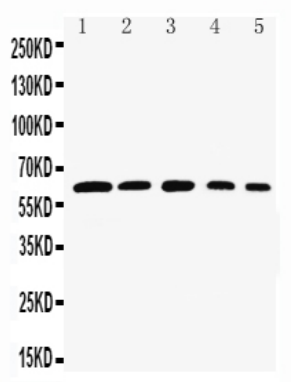
Name	RELB
Function	NF-kappa-B is a pleiotropic transcription factor which is present in almost all cell types and is involved in many biological processed such as inflammation, immunity, differentiation, cell growth, tumorigenesis and apoptosis. NF-kappa-B is a homo- or heterodimeric complex formed by the Rel-like domain-containing proteins RELA/p65, RELB, NFKB1/p105, NFKB1/p50, REL and NFKB2/p52. The dimers bind at kappa-B sites in the DNA of their target genes and the individual dimers have distinct preferences for different kappa-B sites that they can bind with distinguishable affinity and specificity. Different dimer combinations act as transcriptional activators or repressors, respectively. NF-kappa-B is controlled by various mechanisms of post-translational modification and subcellular compartmentalization as well as by interactions with other cofactors or corepressors. NF-kappa-B complexes are held in the cytoplasm in an inactive state complexed with members of the NF-kappa-B inhibitor (I-kappa-B) family. In a conventional activation pathway, I-kappa-B is phosphorylated by I- kappa-B kinases (IKKs) in response to different activators, subsequently degraded thus liberating the active NF-kappa-B complex which translocates to the nucleus. NF-kappa-B heterodimeric RelB-p50 and RelB-p52 complexes are transcriptional activators. RELB neither associates with DNA nor with RELA/p65 or REL. Stimulates promoter activity in the presence of NFKB2/p49. As a member of the NUPR1/RELB/IER3 survival pathway, may provide pancreatic ductal adenocarcinoma with remarkable resistance to cell stress, such as starvation or gemcitabine treatment. Regulates the circadian clock by repressing the transcriptional activator activity of the CLOCK-BMAL1 heterodimer in a CRY1/CRY2 independent manner. Increased repression of the heterodimer is seen in the presence of NFKB2/p52. Is required for both T and B lymphocyte maturation and function (PubMed:26385063).
Cellular Location	Nucleus. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Note=Colocalizes with NEK6 in the centrosome

Background

RELB(v-rel reticuloendotheliosis viral oncogene homolog B) is also known as IREL. The International Radiation Hybrid Mapping Consortium assigned the RELB gene to chromosome 19. By RT-PCR and immunocytochemical analyses, Clark et al.(1999) showed that RELB expression correlated with dendritic cell activation. NF-kappa-B-inducing kinase is required for osteoclastogenesis in response to pathologic stimuli. Vaira et al.(2008) found that overexpression of Relb, but not Rela, rescued differentiation of mouse Nik -/- osteoclast precursors, indicating that blockade of the alternative NF-kappa-B pathway, rather than the classical NF-kappa-B pathway, is responsible for the osteoclastogenic defect in the absence of Nik. Using Relb -/- mice, they showed that Relb itself was required for Rankl-induced osteoclastogenesis in vitro and for TNF-induced bone resorption in vivo. Both Relb -/- and Nik -/- mice were resistant to tumor-mediated osteolysis. Vaira et al.(2008) concluded that the alternative NF-kappa-B pathway, via RELB, plays an essential and unique role in RANKL signaling toward osteoclast development.

Images

Anti-Rel B antibody, ABO11111, Western blottingLane 1: Rat Testis Tissue LysateLane 2: HELA Cell LysateLane 3: NIH3T3 Cell LysateLane 4: RAJI Cell LysateLane 5: HEPA Cell Lysate



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