

RELB Antibody
Purified Mouse Monoclonal Antibody (Mab)
Catalog # AM8565b**Specification**

RELB Antibody - Product Information

Application	WB,E
Primary Accession	Q01201
Reactivity	Human, Mouse
Host	Mouse
Clonality	monoclonal
Isotype	IgG1,k
Calculated MW	62134

RELB Antibody - Additional Information**Gene ID** 5971**Other Names**

Transcription factor RelB, I-Rel, RELB

Target/Specificity

This RELB antibody is generated from a mouse immunized with a recombinant protein human RELB.

Dilution

WB~~1:5000

E~~Use at an assay dependent concentration.

Format

Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

RELB Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

RELB Antibody - 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 processes 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

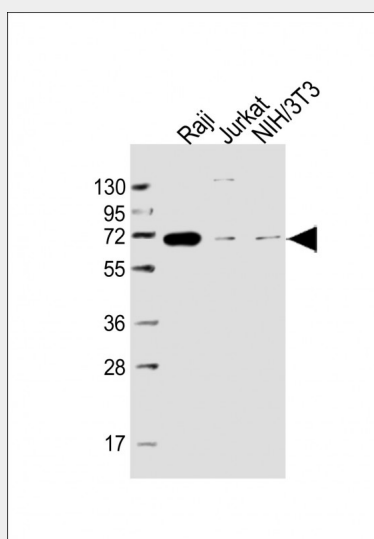
Nucleus. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Note=Colocalizes with NEK6 in the centrosome

RELB Antibody - Protocols

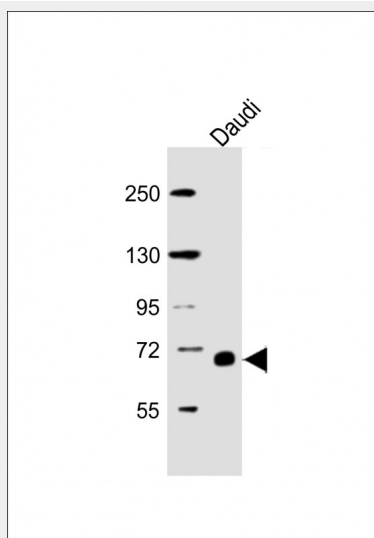
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

RELB Antibody - Images



All lanes : Anti-RELB Antibody at 1:5000 dilution Lane 1: Raji whole cell lysate Lane 2: Jurkat whole cell lysate Lane 3: NIH/3T3 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-mouse IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 62 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



Anti-RELB Antibody at 1:5000 dilution + Daudi whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-mouse IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 62 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

RELB Antibody - Background

NF-kappa-B is a pleiotropic transcription factor which is present in almost all cell types and is involved in many biological processes 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-ARNTL/BMAL1 heterodimer in a CRY1/CRY2 independent manner. Increased repression of the heterodimer is seen in the presence of NFKB2/p52.

RELB Antibody - References

- Ruben S.M., et al. *Genes Dev.* 6:745-760(1992).
- Yoshiura K., et al. Submitted (JAN-1998) to the EMBL/GenBank/DDBJ databases.
- Ryseck R.P., et al. *Mol. Cell. Biol.* 12:674-684(1992).
- Dobrzanski P., et al. *Mol. Cell. Biol.* 13:1572-1582(1993).
- Dobrzanski P., et al. *EMBO J.* 13:4608-4616(1994).

