Anti-RelB Antibody [SD07-39]

ET1612-18

Product Type:	Recombinant Rabbit monoclonal IgG, primary antibodies
Species reactivity:	Human
Applications:	WB, IF-Tissue, IHC-P, IP
Molecular Wt:	Predicted band size: 62 kDa
Clone number:	SD07-39

Description: In resting cells, RelB is sequestered by the NF-κB precursor protein p100 in the cytoplasm. A select set of TNF-R superfamily members, including lymphotoxin β-receptor (LTβR), BAFF-R, CD40 and RANK, activate the non-canonical NF-κB pathway. In this pathway, NIK stimulates the processing of p100 into p52, which in association with RelB appears in the nucleus as RelB:p52 NF-κB heterodimers. RelB:p52 activates the expression homeostatic lymphokines,which instruct lymphoid organogenesis and determine the trafficking of naive lymphocytes in the secondary lymphoid organs. Recent studies has suggested that the functional non-canonical NF-κB pathway is modulated by canonical NF-κB signalling. For example, syntheses of the constituents of the non-canonical pathway, viz RelB and p52, are controlled by canonical IKK2-IκB-RelA:p50 signalling.Moreover, generation of canonical and non-canonical dimers, viz RelA:p50 and RelB:p52, within the cellular milieu are mechanistically interlinked. These analyses suggest that an integrated NF-κB system network underlies activation of both RelA and RelB containing dimer and that a malfunctioning canonical pathway will lead to an aberrant cellular response also through the non-canonical pathway. Most intriguingly, a recent study identified that TNF-induced canonical signalling subverts non-canonical RelB:p52 activity in the inflamed lymphoid tissues limiting lymphocyte ingress.Mechanistically, TNF inactivated NIK in LTβR - stimulated cells and induced the synthesis of Nfkb2 mRNA encoding p100; these together potently accumulated unprocessed p100, which attenuated the RelB activity. A role of p100/Nfkb2 in dictating lymphocyte ingress in the inflamed lymphoid tissue may have broad physiological implications.

Immunogen:	Synthetic peptide within Human RelB aa 1-50 / 579.
Positive control:	Raji cell lysate, Daudi cell lysate, human tonsil tissue.
Subcellular location:	Nucleus, Cytoplasm, cytoskeleton, microtubule organizing center, centrosome.
Database links:	SwissProt: Q01201 Human
Recommended Dilutions:	
WB	1:1,000
IF-Tissue	1:50-1:200
IHC-P	1:50-1:200
IP	Use at an assay dependent concentration.
Storage Buffer:	1*TBS (pH7.4), 0.05% BSA, 40% Glycerol. Preservative: 0.05% Sodium Azide.
Storage Instruction:	Shipped at 4℃. Store at +4℃ short term (1-2 weeks). It is recommended to aliquot into single-use upon delivery. Store at -20℃ long term.
Purity:	Protein A affinity purified.

Images

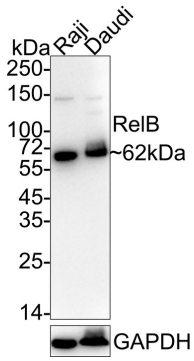

Fig1: Western blot analysis of RelB on different lysates with Rabbit anti-RelB antibody (ET1612-18) at 1/1,000 dilution.

Lane 1: Raji cell lysate
Lane 2: Daudi cell lysate

Lysates/proteins at 20 µg/Lane.

Predicted band size: 62 kDa
Observed band size: 62 kDa

Exposure time: 3 minutes; ECL: K1801;

4-20% SDS-PAGE gel.

Proteins were transferred to a PVDF membrane and blocked with 5% NFDM/TBST for 1 hour at room temperature. The primary antibody (ET1612-18) at 1/1,000 dilution was used in 5% NFDM/TBST at 4℃ overnight. Goat Anti-Rabbit IgG - HRP Secondary Antibody (HA1001) at 1/50,000 dilution was used for 1 hour at room temperature.

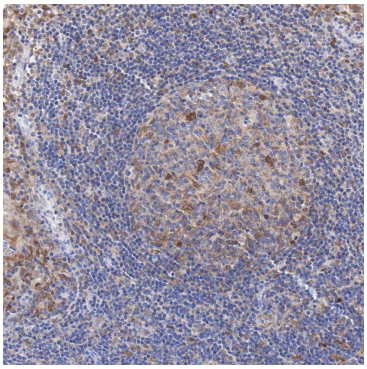

Fig2: Immunohistochemical analysis of paraffin-embedded human tonsil tissue with Rabbit anti-RelB antibody (ET1612-18) at 1/50 dilution.

The section was pre-treated using heat mediated antigen retrieval with sodium citrate buffer (pH 6.0) (high pressure) for 2 minutes. The tissues were blocked in 1% BSA for 20 minutes at room temperature, washed with ddH_2O and PBS, and then probed with the primary antibody (ET1612-18) at 1/50 dilution for 1 hour at room temperature. The detection was performed using an HRP conjugated compact polymer system. DAB was used as the chromogen. Tissues were counterstained with hematoxylin and mounted with DPX.

Background References

1. Siak JJ et al. The nuclear-factor kappaB pathway is activated in pterygium. Invest Ophthalmol Vis Sci 52:230-6 (2011).
2. Huang Y et al. Gene expression profiling identifies emerging oncogenic pathways operating in extranodal NK/T-cell lymphoma, nasal type. Blood 115:1226-37 (2010).