

C-rel Antibody(NFkB) Antibody(C-term D504) Blocking peptide
Synthetic peptide
Catalog # BP6336b**Specification**

C-rel Antibody(NFkB) Antibody(C-term D504) Blocking peptide - Product InformationPrimary Accession [Q04864](#)**C-rel Antibody(NFkB) Antibody(C-term D504) Blocking peptide - Additional Information****Gene ID** 5966**Other Names**

Proto-oncogene c-Rel, REL

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP6336b](/product/products/AP6336b) was selected from the NFkB region of human C-rel (NFkB) (C-term D504). A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

C-rel Antibody(NFkB) Antibody(C-term D504) Blocking peptide - Protein Information**Name** REL**Function**

Proto-oncogene that may play a role in differentiation and lymphopoiesis. 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. The NF-kappa-B heterodimer RELA/p65- c-Rel is a transcriptional activator.

Cellular Location

Nucleus.

C-rel Antibody(NFkB) Antibody(C-term D504) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

C-rel Antibody(NFkB) Antibody(C-term D504) Blocking peptide - Images**C-rel Antibody(NFkB) Antibody(C-term D504) Blocking peptide - Background**

Nuclear factor (NF)-kappa B is a sequence specific transcriptional activator that binds to the intronic enhancer of kappa light chain gene in B lymphocytes. NF-kB regulates the expression of a wide variety of genes that involved in apoptosis, viral life cycle, tumorigenesis, autoimmune diseases and inflammation. NF-kB is a heterodimer of members of the rel family of proteins such as p50, p65, and c-rel. In most cells, inhibitory Ikb proteins sequester NF-kB/Rel in the cytoplasm. Cellular stimulation precipitates degradation of Ikb and modification of NF-kB/Rel proteins, permitting translocation of NF-kB/Rel (c-Rel and RelA) to the nucleus for action on target genes. The important role of c-Rel in B-cell development, growth, and survival has been intensively studied, as well as its function in differentiation and lymphopoiesis (particularly lymphoid cancer).

C-rel Antibody(NFkB) Antibody(C-term D504) Blocking peptide - References

Jain, A., et al., J. Clin. Invest. 114(11):1593-1602 (2004).Xiao, Q., et al., Appl. Immunohistochem. Mol. Morphol. 12(3):211-215 (2004).Houldsworth, J., et al., Blood 103(5):1862-1868 (2004).Phelps, C.B., et al., Oncogene 23(6):1229-1238 (2004).Bernard, D., et al., Cancer Res. 64(2):472-481 (2004).