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Gene Section

Mini Review

RELB (v-rel reticuloendotheliosis viral oncogene homolog B)

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Identity

Other names: I-Rel

HGNC (Hugo): RELB

Location: 19q13.32

Note

See also, in the Deep Insight section: Upstream Signal Transduction of NF-kB Activation.

DNA/RNA

Description

The gene encoding human RelB has 11 exons spanning ~37 kb. Analysis of the 5'-flanking region of human relb gene indicates that RelB transcription is dependent on a TATA-less promoter containing two kB sites. Thus, while relA is constitutively expressed, the expression of both rel and relb is in an inducible fashion and dependent on NF-kB.

Protein

Description

The human relb gene encodes a protein composed 579 amino acids with an approximately molecular weight of 66 kDa. Although structurally similar with other Rel family proteins containing RHD, NLS and TA domain, RelB contains an additional 121 amino acid region located at the N-terminus of RHD. Original study indicated that RelB failed to associate with RelA(p65) and to interact with DNA. That is the reason why it was named as inhibitive-Rel (I-Rel). In contrast, later studies demonstrated that RelB was able to form a heterodimer with NF-kB p50 or p52 and induce the transcription of target constructs or genes. No DNA binding activity has been suggested for the homodimeric complex of RelB, which may be possibly due to the N-terminal 121 amino acid domain that interfered with the DNA binding of RHD.

Expression

Wide.

Localisation

Cytosol, nuclei after activation.

Function

regulation of the genes involved in cell-to-cell interaction, intercellular communication, cell recruitment or transmigration, amplification or spreading of primary pathogenic signals, cell apoptosis, and initiation or acceleration of tumorigenesis. Interaction with:members of IkB family and Rel family.

Implicated in

Cancer, autoimmune arthritis, glomerulonephritis, asthma, inflammatory bowel disease, septic shock, lung fibrosis, HTLV-1 infection, and AIDS

References

Ruben SM, Klement JF, Coleman TA, Maher M, Chen CH, Rosen CA. I-Rel: a novel rel-related protein that inhibits NF-kappa B transcriptional activity. Genes Dev. 1992 May;6(5):745-60

Bours V, Azarenko V, Dejardin E, Siebenlist U. Human RelB (I-Rel) functions as a kappa B site-dependent transactivating member of the family of Rel-related proteins. Oncogene. 1994 Jun;9(6):1699-702

Baldwin AS Jr. The NF-kappa B and I kappa B proteins: new discoveries and insights. Annu Rev Immunol. 1996;14:649-83

Ghosh S, May MJ, Kopp EB. NF-kappa B and Rel proteins: evolutionarily conserved mediators of immune responses. Annu Rev Immunol. 1998;16:225-60

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