

## BCL10

**Reactivity:**Human Mouse Rat

**Tested applications:**WB IHC IF

**Recommended Dilution:**WB 1:500 - 1:2000 IHC 1:50 - 1:200 IF 1:50 - 1:200

**Calculated MW:**26kDa

**Observed MW:**Refer to Figures

**Immunogen:**

Recombinant protein of human BCL10

**Storage Buffer:**

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

**Concentration:**

b

**Synonym:**

BCL10;CARMEN;CIPER;CLAP;c-E10;mE10 ;

**Catalog #:**A1106

**Antibody Type:**

Polyclonal Antibody

**Species:**Rabbit

**Gene ID:**8915

**Isotype:**IgG

**Swiss Prot:**O95999

**Purity:**Affinity purification

For research use only.

**Background:**

Bcl10/CIPER/CLAP/mE10 is a widely expressed CARD (caspase recruitment domain) containing protein shown to induce apoptosis and activate NF- $\kappa$ B (1-5). The CARD domain mediates self-oligomerization, interactions with other CARD proteins and is necessary for NF- $\kappa$ B activation, although the precise mechanism which Bcl10 regulates these processes is not fully understood. The discovery of Bcl10 came from observations of the chromosomal translocation t(1;14)(p22;q32) from B cell lymphomas of the mucosa-associated lymphoid tissue (MALT) (1,5). This translocation results in deregulated expression of a truncated form of Bcl10 which lacks apoptotic activity and enhances transformation. Studies from Bcl10 deficient mice demonstrate that Bcl10 is essential for the activation of NF- $\kappa$ B by T- and B-cell receptors (6). One third of Bcl10 deficient mice developed lethal exencephaly. Surviving mice were unaffected by various apoptotic stimuli, but were severely immunodeficient and defective in antigen receptor-induced NF- $\kappa$ B activation. PKC or T-cell receptor signaling results in a downregulation of Bcl10 protein levels, attenuating both NF- $\kappa$ B activation and cellular proliferation and also provides a negative feedback regulation of the NF- $\kappa$ B signaling to T cell signaling (7).

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