## Bcl10 (C78F1) Rabbit mAb





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Applications: W, IP	<b>Reactivity:</b> H M R	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 28	<b>Source/Isotype:</b> Rabbit IgG	<b>UniProt ID:</b> #O95999	Entrez-Gene Id: 8915		
Product Usage Information		<b>Application</b> Western Blotting Immunoprecipitation			<b>Dilution</b> 1:1000 1:50			
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.						
Specificity/Sen	sitivity	Bcl10 (C78F1) Rabbit mAb detects endogenous levels of total Bcl10 protein.						
Species predict based on 100% homology		Monkey						
Source / Purific	cation	Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Ser60 of Bcl10.						
Background		Bcl10/CIPER/CLAP/mE10 is a widely expressed CARD (caspase recruitment domain) containing protein shown to induce apoptosis and activate NF-κB (1-5). The CARD domain mediates self-oligomerization, interactions with other CARD proteins and is necessary for NF-κB 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-κB 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-κB activiation. PKC or T-cell receptor signaling results in a downregulation of Bcl10 protein levels, attenuating both NF-κB activation and cellular proliferation and also provides a negative feedback regulation of the NF-κB signaling to T cell signaling (7).						
Background Re	eferences	<ol> <li>Willis, T.G. et al. (1999) <i>Cell</i> 96, 35-45.</li> <li>Koseki, T. et al. (1999) <i>J Biol Chem</i> 274, 9955-61.</li> <li>Srinivasula, S.M. et al. (1999) <i>J Biol Chem</i> 274, 17946-54.</li> <li>Yan, M. et al. (1999) <i>J Biol Chem</i> 274, 10287-92.</li> <li>Zhang, Q. et al. (1999) <i>Nat Genet</i> 22, 63-8.</li> <li>Ruland, J. et al. (2001) <i>Cell</i> 104, 33-42.</li> <li>Scharschmidt, E. et al. (2004) <i>Mol Cell Biol</i> 24, 3860-73.</li> </ol>						
Species Reactiv	vitv	Species reactivity is de	termined by testin	n in at least one approve	ad application (e.g.	western blot)		
•	-	Species reactivity is determined by testing in at least one approved application (e.g., western blot).						
Western Blot B	Suffer		PORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X 5, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.					
Applications K	ey	W: Western Blotting IP: Immunoprecipitation						
Cross-Reactivit	y Key	H: Human M: Mouse R: Rat						
Trademarks an	d Patents	Cell Signaling Technology is a trademark of Cell Signaling Technology, Inc.						
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