

Anti-BCL6 Antibody (Center)

Catalog Number: M00142

About BCL6

Transcriptional repressor mainly required for germinal center (GC) formation and antibody affinity maturation which has different mechanisms of action specific to the lineage and biological functions. Forms complexes with different corepressors and histone deacetylases to repress the transcriptional expression of different subsets of target genes. Represses its target genes by binding directly to the DNA sequence 5'-TTCCTAGAA-3' (BCL6- binding site) or indirectly by repressing the transcriptional activity of transcription factors. In GC B-cells, represses genes that function in differentiation, inflammation, apoptosis and cell cycle control, also autoregulates its transcriptional expression and up-regulates, indirectly, the expression of some genes important for GC reactions, such as AICDA, through the repression of microRNAs expression, like miR155. An important function is to allow GC B-cells to proliferate very rapidly in response to T-cell dependent antigens and tolerate the physiological DNA breaks required for immunoglobulin class switch recombination and somatic hypermutation without inducing a p53/TP53-dependent apoptotic response. In follicular helper CD4(+) T-cells (T(FH) cells), promotes the expression of T(FH)-related genes but inhibits the differentiation of T(H)1, T(H)2 and T(H)17 cells. Also required for the establishment and maintenance of immunological memory for both T- and B-cells. Suppresses macrophage proliferation through competition with STAT5 for STAT-binding motifs binding on certain target genes, such as CCL2 and CCND2. In response to genotoxic stress, controls cell cycle arrest in GC B-cells in both p53/TP53- dependent and -independent manners. Besides, also controls neurogenesis through the alteration of the composition of NOTCH- dependent transcriptional complexes at selective NOTCH targets, such as HES5, including the recruitment of the deacetylase SIRT1 and resulting in an epigenetic silencing leading to neuronal differentiation.

Overview

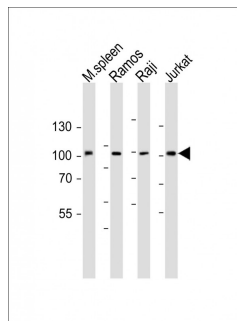
Product Name	Anti-BCL6 Antibody (Center)
Reactive Species	Human, Mouse
Description	Boster Bio Anti-BCL6 Antibody (Center) (Catalog # M00142). Tested in WB, IHC-P application(s). This antibody reacts with Human, Mouse.
Application	IHC-P, WB
Clonality	Polyclonal
Formulation	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide.
Storage Instructions	Maintain refrigerated at 2-8°C for up to 2 weeks. For long-term storage, store at -20°C in small aliquots to prevent freeze-thaw cycles.
Host	Rabbit
Uniprot ID	P41182

Technical Details

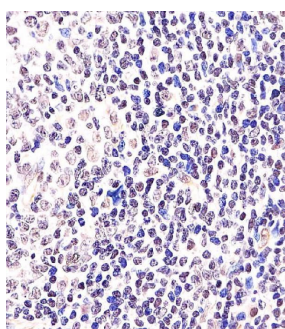
Immunogen	This BCL6 antibody is generated from a rabbit immunized with a KLH conjugated synthetic peptide between 364-395 amino acids from the Central region of human BCL6.
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Predicted Reactive Species	Human
Isotype	Rabbit IgG
Purification	This antibody is purified through a protein A column, followed by peptide affinity purification.
Suggested Dilutions	WB: 1:1000 IHC-P: 1:25

Anti-BCL6 Antibody (Center) (M00142) Images



All lanes : Anti-BCL6 Antibody (Center) at 1:1000 dilution
Lane 1: mouse spleen lysate
Lane 2: Ramos whole cell lysate
Lane 3: Raji whole cell lysate
Lane 4: Jurkat whole cell lysate
Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 79 kDa
Blocking/Dilution buffer: 5% NFDM/TBST.



M00142 staining BCL6 in Human tonsil tissue sections by Immunohistochemistry (IHC-P -paraformaldehyde-fixed, paraffin-embedded sections). Tissue was fixed with formaldehyde and blocked with 3% BSA for 0.5 hour at room temperature; antigen retrieval was by heat mediation with a citrate buffer (pH6). Samples were incubated with primary antibody (1/25) for 1 hours at 37°C. A undiluted biotinylated goat polyvalent antibody was used as the secondary antibody.

1 Publications Citing This Product

1. PubMed ID: 10.1016/j.molimm.2020.06.003, An imbalance between blood CD4+CXCR5+Foxp3+ Tfr cells and CD4+CXCR5+Tfh cells may contribute to the immunopathogenesis of rheumatoid arthritis

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