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DATA SHEET

Mouse Anti-Bcl-10 [SPM520]: MC0612, MC0612RTU7

Intended Use: For Research Use Only

Description: Bcl-10, with an N-terminal caspase recruitment domain (CARD), is found in a number of apoptotic regulatory molecules. It was identified through its direct involvement in t(1;14) of mucosa-associated lymphoid tissue (MALT) lymphoma. Expression of BCL10 was shown to induce NFKB activation in a NIK-dependent pathway. This antibody labels subpopulations of normal B and T cells and is a useful tool for the sub-classification of lymphomas. In MALT lymphomas with the t(1;14) translocation, while 55% of MALT lymphomas lacking this translocation exhibited the same labeling pattern, although at a much lower level.

Specifications:

Clone:	SPM520
Source:	Mouse
Isotype:	IgG1k
Reactivity:	Human
Immunogen:	Human BCL10 recombinant protein aa122-168
Localization:	Cytoplasm, nucleus
Formulation:	Purified antibody in PBS pH7.4, containing BSA and ≤ 0.09% sodium azide (NaN3)
Storage:	Store at 2°- 8°C
Applications:	IHC, Flow Cyt., ICC/IF, WB

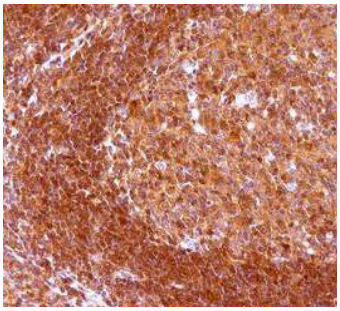
Package:

Description	Catalog No.	Size
Bcl-10 Concentrated	MC0612	1 ml
Bcl-10 Prediluted	MC0612RTU7	7 ml

IHC Procedure*:

Positive Control Tissue:	Lymphoma, WEHI-231, Ramos cells
Concentrated Dilution:	50-200
Pretreatment:	Tris EDTA pH9.0, 15 minutes Pressure Cooker or 30-60 minutes water bath at 95°-99°C
Incubation Time and Temp:	30-60 minutes @ RT
Detection:	Refer to the detection system manual

* Result should be confirmed by an established diagnostic procedure.



FFPE human tonsil stained with anti-Bcl-10 using DAB

References:

1. Two Antagonistic MALT1 Auto-Cleavage Mechanisms Reveal a Role for TRAF6 to Unleash MALT1 Activation. Ginster S, et al. PLoS One 12:e0169026, 2017.
2. Alternative splicing of MALT1 controls signalling and activation of CD4(+) T cells. Meininger I, et al. Nat Commun 7:11292, 2016.
3. Pharmacological inhibition of MALT1 protease activity protects mice in a mouse model of multiple sclerosis. Mc Guire C, et al. J Neuroinflammation 11:124, 2014.

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