

Bcl-2 (Apoptosis & Follicular Lymphoma Marker) Antibody - With BSA and Azide
Mouse Monoclonal Antibody [Clone SPM117]
Catalog # AH10717

Specification

**Bcl-2 (Apoptosis & Follicular Lymphoma Marker) Antibody - With BSA and Azide -
Product Information**

Application	,1,14,3,4,
Primary Accession	P10415
Other Accession	596 , 150749
Reactivity	Human
Host	Mouse
Clonality	Monoclonal
Isotype	Mouse / IgG1, kappa
Calculated MW	25-26kDa KDa

**Bcl-2 (Apoptosis & Follicular Lymphoma Marker) Antibody - With BSA and Azide -
Additional Information**

Gene ID 596

Other Names

Apoptosis regulator Bcl-2, BCL2

Format

200ug/ml of Ab purified from Bioreactor Concentrate by Protein A/G. Prepared in 10mM PBS with 0.05% BSA & 0.05% azide. Also available WITHOUT BSA & azide at 1.0mg/ml.

Storage

Store at 2 to 8°C. Antibody is stable for 24 months.

Precautions

Bcl-2 (Apoptosis & Follicular Lymphoma Marker) Antibody - With BSA and Azide is for research use only and not for use in diagnostic or therapeutic procedures.

**Bcl-2 (Apoptosis & Follicular Lymphoma Marker) Antibody - With BSA and Azide -
Protein Information**

Name BCL2

Function

Suppresses apoptosis in a variety of cell systems including factor-dependent lymphohematopoietic and neural cells (PubMed: [1508712](http://www.uniprot.org/citations/1508712), PubMed: [8183370](http://www.uniprot.org/citations/8183370)). Regulates cell death by controlling the mitochondrial membrane permeability (PubMed: [11368354](http://www.uniprot.org/citations/11368354)). Appears to function in a feedback loop system with caspases (PubMed: [11368354](http://www.uniprot.org/citations/11368354)).

Inhibits caspase activity either by preventing the release of cytochrome c from the mitochondria and/or by binding to the apoptosis-activating factor (APAF-1) (PubMed:11368354). Also acts as an inhibitor of autophagy: interacts with BECN1 and AMBRA1 during non-starvation conditions and inhibits their autophagy function (PubMed:18570871, PubMed:21358617, PubMed:20889974). May attenuate inflammation by impairing NLRP1-inflammasome activation, hence CASP1 activation and IL1B release (PubMed:17418785).

Cellular Location

Mitochondrion outer membrane; Single-pass membrane protein. Nucleus membrane; Single-pass membrane protein. Endoplasmic reticulum membrane; Single-pass membrane protein. Cytoplasm {ECO:0000250|UniProtKB:P10417}

Tissue Location

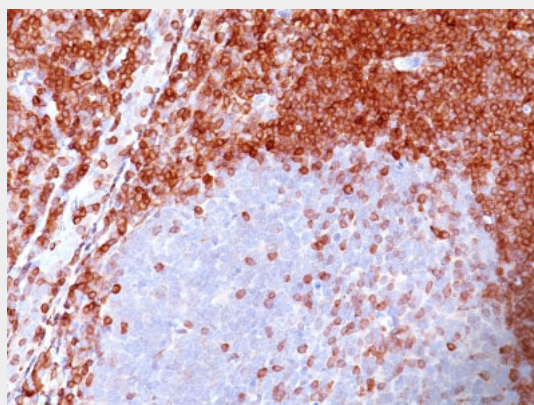
Expressed in a variety of tissues.

Bcl-2 (Apoptosis & Follicular Lymphoma Marker) Antibody - With BSA and Azide - Protocols

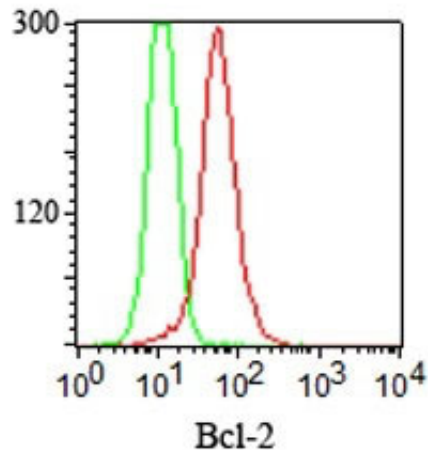
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

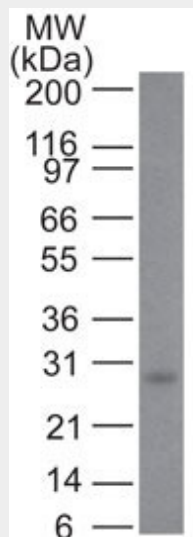
Bcl-2 (Apoptosis & Follicular Lymphoma Marker) Antibody - With BSA and Azide - Images



Formalin-fixed, paraffin-embedded human Non-Hodgkin's Lymphoma stained with Bcl-2 Ab (SPM117).



Flow Cytometry of Jurkat cells using Bcl-2 Ab (SPM117) (red) and isotype control (green).



Western Blot of Bcl-2 in human Skin using Bcl-2 Ab (SPM117).

Bcl-2 (Apoptosis & Follicular Lymphoma Marker) Antibody - With BSA and Azide - Background

This antibody recognizes a protein of 25-26kDa, identified as the bcl-2 α oncoprotein. It shows no cross-reaction with Bcl-x or Bax protein. Expression of bcl-2 α oncoprotein inhibits the programmed cell death (apoptosis). In most follicular lymphomas, neoplastic germinal centers express high levels of bcl-2 α protein, whereas the normal or hyperplastic germinal centers are negative. Consequently, this antibody is valuable when distinguishing between reactive and neoplastic follicular proliferation in lymph node biopsies. It may also be used in distinguishing between those follicular lymphomas that express bcl-2 protein and the small number in which the neoplastic cells are bcl-2 negative.

Bcl-2 (Apoptosis & Follicular Lymphoma Marker) Antibody - With BSA and Azide - References

Pezzella F et. al.. American Journal of Pathology, 1990, 137(2):225-32