

BCL2 antibody - N-terminal region
Rabbit Polyclonal Antibody
Catalog # AI16168**Specification**

BCL2 antibody - N-terminal region - Product Information

Application	WB, IHC
Primary Accession	P10415
Other Accession	NM_000633 , NP_000624
Reactivity	Human
Predicted	Human
Host	Rabbit
Clonality	Polyclonal
Calculated MW	26kDa kDa

BCL2 antibody - N-terminal region - Additional Information**Gene ID 596**

Alias Symbol	Bcl-2, PPP1R50
Other Names	
Apoptosis regulator Bcl-2, BCL2	

Format

Liquid. Purified antibody supplied in 1x PBS buffer with 0.09% (w/v) sodium azide and 2% sucrose.

Reconstitution & Storage

Add 50 ul of distilled water. Final anti-BCL2 antibody concentration is 1 mg/ml in PBS buffer with 2% sucrose. For longer periods of storage, store at 20°C. Avoid repeat freeze-thaw cycles.

Precautions

BCL2 antibody - N-terminal region is for research use only and not for use in diagnostic or therapeutic procedures.

BCL2 antibody - N-terminal region - Protein Information**Name BCL2****Function**

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

[11368354](http://www.uniprot.org/citations/11368354)). Also acts as an inhibitor of autophagy: interacts with BECN1 and AMBRA1 during non-starvation conditions and inhibits their autophagy function (PubMed: [18570871](http://www.uniprot.org/citations/18570871), PubMed: [20889974](http://www.uniprot.org/citations/20889974), PubMed: [21358617](http://www.uniprot.org/citations/21358617)). May attenuate inflammation by impairing NLRP1-inflammasome activation, hence CASP1 activation and IL1B release (PubMed: [17418785](http://www.uniprot.org/citations/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

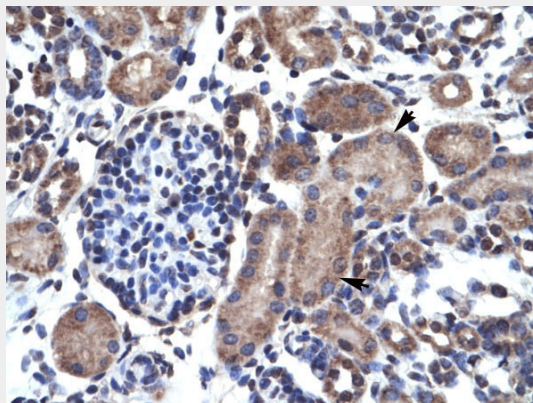
Expressed in a variety of tissues.

BCL2 antibody - N-terminal region - Protocols

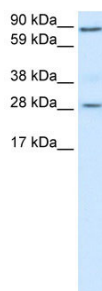
Provided below are standard protocols that you may find useful for product applications.

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

BCL2 antibody - N-terminal region - Images



Human kidney



90 kDa
59 kDa
38 kDa
28 kDa
17 kDa

WB Suggested Anti-BCL2 Antibody Titration: 0.2-1 µg/ml
Positive Control: Human Placenta

BCL2 antibody - N-terminal region - Background

Suppresses apoptosis in a variety of cell systems including factor-dependent lymphohematopoietic and neural cells. Regulates cell death by controlling the mitochondrial membrane permeability. Appears to function in a feedback loop system with caspases. 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).

BCL2 antibody - N-terminal region - References

Tsujimoto Y., et al. Proc. Natl. Acad. Sci. U.S.A. 83:5214-5218(1986).
Eguchi Y., et al. Nucleic Acids Res. 20:4187-4192(1992).
Cleary M.L., et al. Cell 47:19-28(1986).
Seto M., et al. EMBO J. 7:123-131(1988).
Hua C., et al. Oncogene Res. 2:263-275(1988).