

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

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**BCL2 antibody - N-terminal region - Product Information**

Application	<b>WB, IHC</b>
Primary Accession	<a href="#">P10415</a>
Other Accession	<a href="#">NM_000633</a> , <a href="#">NP_000624</a>
Reactivity	<b>Human</b>
Predicted	<b>Human</b>
Host	<b>Rabbit</b>
Clonality	<b>Polyclonal</b>
Calculated MW	<b>26kDa KDa</b>

**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</a>, PubMed:<a href="http://www.uniprot.org/citations/8183370" target="\_blank">8183370</a>). Regulates cell death by controlling the mitochondrial membrane permeability (PubMed:<a href="http://www.uniprot.org/citations/11368354" target="\_blank">11368354</a>). Appears to function in a feedback loop system with caspases (PubMed:<a href="http://www.uniprot.org/citations/11368354" target="\_blank">11368354</a>). 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

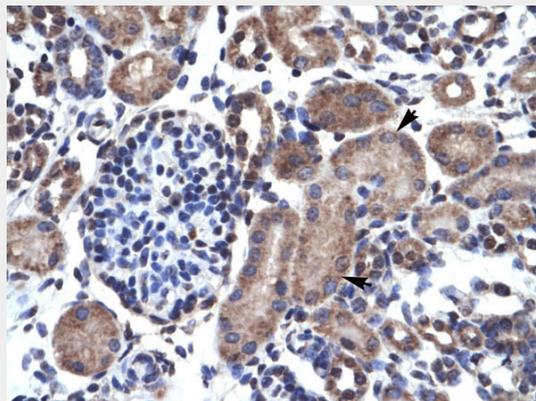
Expressed in a variety of tissues.

### BCL2 antibody - N-terminal region - Protocols

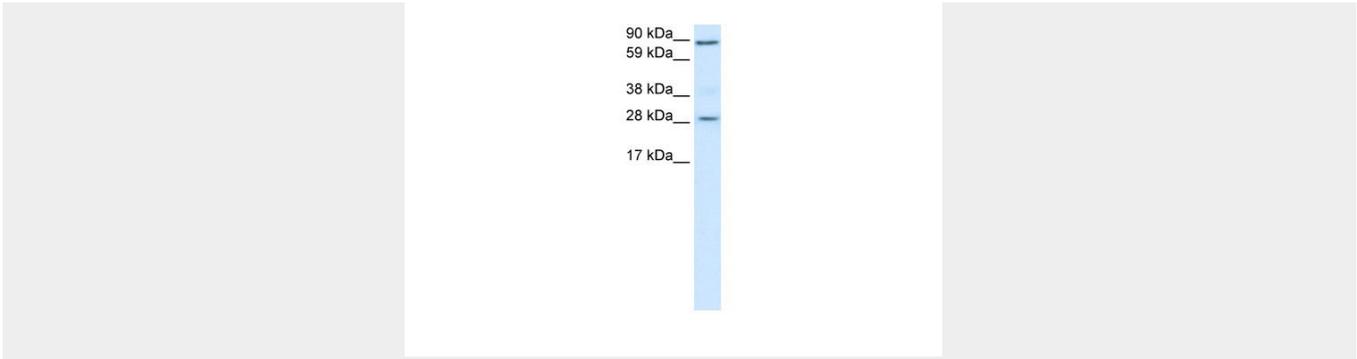
Provided below are standard protocols that you may find useful for product applications.

- [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).