

Rabbit Anti-Phospho-Bcl-2 (Ser70)/Cy5 Conjugated antibody

SL3052R-Cy5

Product Name	Anti-Phospho-Bcl-2 (Ser70)/Cy5
Chinese Name	Cy5 标记的磷酸化 Bcl-2 抗体
Alias	Bcl2 (phospho S70); p-Bcl2 (phospho S70); Apoptosis regulator Bcl 2; Apoptosis regulator Bcl2; AW986256; B cell CLL/lymphoma 2; B cell leukemia/lymphoma 2; B cell lymphoma 2; Bcl 2; Bcl-2; Bcl2; BCL2 protein; C430015F12Rik; D630044D05Rik; D830018M01Rik; Leukemia/lymphoma, B-cell, 2; Oncogene B-cell leukemia 2; BCL2_HUMAN.
Product Type	Phosphorylated anti
Research Area	Tumour Cell biology Neurobiology Signal transduction Apoptosis Mitochondrion
Immunogen Species	Rabbit
Clonality	Polyclonal
React Species	Human(predicted:Mouse,Rat,Dog,Rabbit)
Applications	Flow-Cyt=1ug/test,IF=1:100-500 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight	26kDa
Form	Lyophilized or Liquid
Concentration	1mg/ml
immunogen	KLH conjugated Synthesised phosphopeptide derived from rat Bcl-2 around the phosphorylation site of Ser70
Lsotype	IgG
Purification	affinity purified by Protein A
Storage Buffer	1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Storage	

background:

BCL2 is an integral outer mitochondrial membrane protein that blocks the apoptotic death of some cells such as lymphocytes. Constitutive expression of BCL2, such as in the case of translocation of BCL2 to Ig heavy chain locus, is thought to be the cause of follicular lymphoma. Two transcript variants (alpha and beta) produced by alternate splicing, differ in their C-terminal ends. BCL2 suppresses apoptosis in a variety of cell systems including factor-dependent lymphohematopoietic and neural cells. It regulates cell death by controlling the mitochondrial membrane permeability. It appears to function in a feedback loop system with caspases. BCL2 inhibits caspase activity either by preventing the release of cytochrome c from the mitochondria and/or by binding to the apoptosis-activating factor (APAF1). It can form homodimers, and heterodimers with BAX, BAD, BAK and BclX(L). Heterodimerization with BAX requires intact BH1 and BH2 domains, and is necessary for anti-apoptotic activity. Also interacts with APAF1, RAF1, TP53BP2, BBC3, BCL2L1 and BNIPL.

Function:

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).

Product Detail

Subunit:

Forms homodimers, and heterodimers with BAX, BAD, BAK and Bcl-X(L). Heterodimerization with BAX requires intact BH1 and BH2 motifs, and is necessary for anti-apoptotic activity. Interacts with EI24. Also interacts with APAF1, BBC3, BCL2L1, BNIPL, MRPL41 and TP53BP2. Binding to FKBP8 seems to target BCL2 to the mitochondria and probably interferes with the binding of BCL2 to its targets. Interacts with BAG1 in an ATP-dependent manner. Interacts with RAF1 (the 'Ser-338' and 'Ser-339' phosphorylated form). Interacts (via the BH4 domain) with EGLN3; the interaction prevents the formation of the BAX-BCL2 complex and inhibits the anti-apoptotic activity of BCL2. Interacts with G0S2; this interaction also prevents the formation of the anti-apoptotic BAX-BCL2 complex.

Subcellular Location:

Mitochondrion outer membrane; Single-pass membrane protein. Nucleus membrane; Single-pass membrane protein. Endoplasmic reticulum membrane; Single-pass membrane protein.

Tissue Specificity:

Expressed in a variety of tissues.

Post-translational modifications:

Phosphorylation/dephosphorylation on Ser-70 regulates anti-apoptotic activity. Growth factor-stimulated phosphorylation on Ser-70 by PKC is required for the anti-apoptosis activity and occurs during the G2/M phase of the cell cycle. In the absence of growth factors, BCL2 appears to be phosphorylated by other protein kinases such as ERKs and stress-activated kinases. Phosphorylated by MAPK8/JNK1 at Thr-69, Ser-70 and Ser-87, which stimulates starvation-induced autophagy. Dephosphorylated by protein phosphatase 2A (PP2A).

Proteolytically cleaved by caspases during apoptosis. The cleaved protein, lacking the BH4 motif, has pro-apoptotic activity, causes the release of cytochrome c into the cytosol promoting further caspase activity.

Monoubiquitinated by PARK2, leading to increase its stability.

DISEASE:

Note=A chromosomal aberration involving BCL2 has been found in chronic lymphatic leukemia. Translocation t(14;18)(q32;q21) with immunoglobulin gene regions. BCL2 mutations found in non-Hodgkin lymphomas carrying the chromosomal translocation could be attributed to the Ig somatic hypermutation mechanism resulting in nucleotide transitions.

Similarity:

Belongs to the Bcl-2 family.

Database links:

[Entrez Gene: 596](#) Human

[Entrez Gene: 12043](#) Mouse

[Entrez Gene: 24224](#) Rat

[Omim: 151430](#) Human

[SwissProt: O02718](#) Cow

[SwissProt: P10415](#) Human

[SwissProt: P10417](#) Mouse

[SwissProt: P49950](#) Rat



[Unigene: 150749](#) Human

[Unigene: 257460](#) Mouse

[Unigene: 9996](#) Rat

Important Note:

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.