## SANTA CRUZ BIOTECHNOLOGY, INC.

# p-Bcl-2 (74.Ser 70): sc-135757



## BACKGROUND

Apoptosis defines a set of cascades which, when initiated, programs the cell to undergo lethal changes such as membrane blebbing, mitochondrial break down and DNA fragmentation. Bcl-2 is one among many key regulators of apoptosis which are essential for proper development, tissue homeostasis and protection against foreign pathogens. Human Bcl-2 is a membrane-associated, anti-apoptotic oncoprotein that can promote cell survival through protein-protein interactions with other Bcl-2 related family members, such as the death suppressors Bcl-x<sub>L</sub>, Mcl-1, Bcl-w and A1, or the death agonists Bax, Bak, Bik, Bad and BID. The anti-apoptotic function of Bcl-2 can also be regulated through proteolytic processing and phosphorylation. Bcl-2 may promote cell survival by interfering with the activation of the cytochrome c/Apaf-1 pathway through stabilization of the mitochondrial membrane. Mutations in the Bcl-2 gene can contribute to cancers where normal physiological cell death mechanisms are compromised by deregulation of the anti-apoptotic influence of Bcl-2.

## REFERENCES

- Kerr, J.F., et al. 1972. Apoptosis: a basic biological phenomenon with wide-ranging implications in tissue kinetics. Br. J. Cancer 26: 239-257.
- Hockenbery, D., et al. 1990. Bcl-2 is an inner mitochondrial membrane protein that blocks programmed cell death. Nature 348: 334-336.
- Alnemri, E.S., et al. 1992. Overexpressed full-length human Bcl-2 extends the survival of baculovirus-infected Sf9 insect cells. Proc. Natl. Acad. Sci. USA 89: 7295-7299.
- 4. Reed, J.C. 1994. Bcl-2 and the regulation of programmed cell death. J. Cell Biol. 124: 1-6.
- Haldar, S., et al. 1995. Inactivation of Bcl-2 by phosphorylation. Proc. Natl. Acad. Sci. USA 92: 4507-4511.
- Yang, J., et al. 1997. Prevention of apoptosis by Bcl-2: release of cytochrome c from mitochondria blocked. Science 275: 1129-1132.
- Adams, J.M. and Cory, S. 1998. The Bcl-2 protein family: arbiters of cell survival. Science 281: 1322-1326.

## CHROMOSOMAL LOCATION

Genetic locus: BCL2 (human) mapping to 18q21.33, Bcl2 (mouse) mapping to 1 E2.1.

## SOURCE

p-Bcl-2 (74.Ser 70) is a rabbit monoclonal antibody raised against a short amino acid sequence containing Ser 70 phosphorylated Bcl-2 of human origin.

#### PRODUCT

Each vial contains 200  $\mu g~lg G_{2a}$  in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

## **STORAGE**

Store at 4° C, \*\*D0 NOT FREEZE\*\*. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

#### **APPLICATIONS**

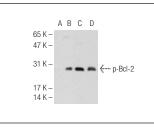
p-Bcl-2 (74.Ser 70) is recommended for detection of Ser 70 phosphorylated Bcl-2 of mouse, rat and human by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunohistochemistry (including paraffinembedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

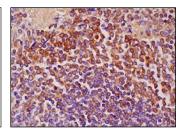
Suitable for use as control antibody for Bcl-2 siRNA (h): sc-29214, Bcl-2 siRNA (m): sc-29215, Bcl-2 shRNA Plasmid (h): sc-29214-SH, Bcl-2 shRNA Plasmid (m): sc-29215-SH, Bcl-2 shRNA (h) Lentiviral Particles: sc-29214-V and Bcl-2 shRNA (m) Lentiviral Particles: sc-29215-V.

Molecular Weight of p-Bcl-2: 26 kDa.

Positive Controls: paclitaxel treated Jurkat whole cell lysate.

#### DATA





Western blot analysis of Bcl-2 phosphorylation in untreated (**A**,**C**), and paclitaxel treated (**B**,**D**) Jurkat whole cell lysates. Antibodies tested include p-Bcl-2 (74.Ser 70): sc-135757 (**A**,**B**) and Bcl-2 (C-2): sc-7382 (**C**,**D**). p-Bcl-2 (74.Ser 70): sc-135757. Immunoperoxidase staining of formalin fixed, paraffin-embedded human spleen tissue showing cytoplasmic staining of cells in white pulp.

## SELECT PRODUCT CITATIONS

- 1. Song, T., et al. 2013. S1 kills MCF-7/ADR cells more than MCF-7 cells: A protective mechanism of endoplasmic reticulum stress. Biomed. Pharmacother. 67: 731-736.
- 2. Acikgoz, E., et al. 2015. Enhanced  $G_2/M$  arrest, caspase related apoptosis and reduced E-Cadherin dependent intercellular adhesion by trabectedin in prostate cancer stem cells. PLoS ONE 10: e0141090.

## **RESEARCH USE**

For research use only, not for use in diagnostic procedures. Not for resale.

#### PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.