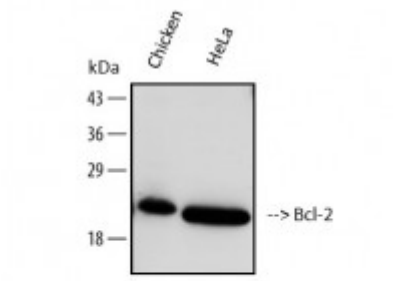
Anti-Bcl-2 antibody



Western Blot (WB) analysis of HeLa and chicken cell using Bcl-2 Antibody (STJ96943), diluted at 1:1000.

Description	Bcl-2 is a protein encoded by the BCL2 gene which is approximately 26,2 kDa. Bcl-2 is localised to the mitochondrion outer membrane, endoplasmic reticulum membrane and nucleus membrane. It is involved in PEDF induced signalling, the TGF-Beta pathway and TNFR1 pathway. It 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 and appears to function in a feedback loop system with caspases. It inhibits caspase activity either by preventing the release of cytochrome C from the mitochondria and/or by binding to the apoptosis-activating factor. Bcl-2 is expressed in a variety of human tissues. Mutations in the BCL2 gene may result in follicular lymphoma. STJ96943 was affinity-purified from mouse ascites by affinity-chromatography using specific immunogen. This antibody detects endogenous Bcl-2 proteins.

Model	STJ96943
Host	Mouse
Reactivity	Human, Mouse, Rat
Applications	IHC, WB
Immunogen	Synthetic Peptide
Gene ID	596
Gene Symbol	BCL2
Dilution range	WB 1:1000-2000IHC 1:200
Specificity	The antibody detects endogenous Bcl-2 proteins.

Tissue Specificity	Expressed in a variety of tissues.
Purification	The antibody was affinity-purified from mouse ascites by affinity-chromatography using specific immunogen.
Clone ID	6B5
Note	For Research Use Only (RUO).
Protein Name	Apoptosis regulator Bcl-2
Clonality	Monoclonal
Conjugation	Unconjugated
Isotype	IgG1
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:990OMIM:151430
Alternative Names	Apoptosis regulator Bcl-2
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). May attenuate inflammation by impairing NLRP1-inflammasome activation, hence CASP1 activation and IL1B release .
Sequence and Domain Family	BH1 and BH2 domains are required for the interaction with BAX and for anti-apoptotic activity. The BH4 motif is required for anti-apoptotic activity and for interaction with RAF1 and EGLN3.; The loop between motifs BH4 and BH3 is required for the interaction with NLRP1.
Cellular Localization	Mitochondrion outer membrane Nucleus membrane Endoplasmic reticulum membrane
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, wich 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 PRKN, leading to increase its stability. Ubiquitinated by SCF(FBXO10), leading to its degradation by the proteasome.