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Phospho-Bcl-2 (Thr56) Rabbit Polyclonal Antibody

Catalog #: EAB10406

Host/Isotype	Clonality	Applications	MW (kDa)	Reactivity
Rabbit IgG	Polyclonal	WB, IP, IHC-P, IF/ICC, ELISA	26	Human, Mouse, Rat

Applications Dilutions

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

WB(Western Blotting)	1:500-2000
IP(Immunoprecipitation)	1:20-200
IHC-P(Immunohistochemistry-Paraffin)	1:50-300
IF/ICC(Immunofluorescence/Immunocytochemistry)	1:50-300
ELISA(Enzyme-linked Immunosorbent Assay)	1:5000-20000

Product Information

Conjugate	Unconjugate
Specificity	Phospho-Bcl-2 (Thr56) Rabbit Polyclonal Antibody detects endogenous levels of Bcl-2 only when phosphorylated at Thr56.
Purification	Affinity purification
Concentration	1mg/ml
Format	Liquid
Formulation	In PBS, pH 7.4, Containing 0.02% sodium azide, 0.5% BSA and 50% Glycerol
Shipping	Gel Pack
Storage	Store at -20°C least 1 year from the date of shipment. Avoid repeated freeze/thaw cycles. Aliquots may be stored at +4°C for 1-2 weeks
UniProt ID	P10415
Entrez-Gene Id	596

Product Descriotion

Bcl-2 is one among many key regulators of apoptosis, which are essential for proper development, tissue homeostasis, and protection against foreign pathogens. Immunostaining of human tissues using the Bcl-2 antibody shows cytoplasmic and membrane staining, as human Bcl-2 is an anti-apoptotic, membrane-associated oncoprotein. The Bcl-2 protein promotes cell survival through protein-protein interactions with other Bcl-2 related family members, such as the death suppressors Bcl-xL, 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.