

<b>Cat. No:</b>	AB-10227
<b>Conjugate:</b>	Unconjugated
<b>Size:</b>	100 ug
<b>Clone:</b>	POLY
<b>Concentration:</b>	1mg/ml
<b>Host:</b>	Rb
<b>Isotype:</b>	IgG
<b>Immunogen:</b>	Peptide derived from the N-terminal sequence of human bcl2. Antibody recognizes the epitope located between Ala42 - Ala60.
<b>Reactivity:</b>	Hu
<b>Applications:</b>	WB: 1:5,000 ELISA - 1:50,000 - 1:100,000 ICC - 1:100 - 1:300 IP: 1:100 - 1:300
<b>Purification:</b>	Aff. Pur.

**Background:** Bcl-2 family of proteins is a key regulator of apoptosis that function to either inhibit or promote cell death. The over expression of members such as Bcl-2 and Bcl-xL inhibit the apoptotic process (1,2). The Bcl-2 family members are also characterized by dimerizing to further modulate apoptosis. Bag-1, for example, has been found to form a heterodimer with Bcl-2 resulting in the enhancement of the anti-apoptotic effect of Bcl-2 (3,4). Other anti-apoptotic Bcl-2 family members include A1, Bcl-xg, Bcl-xb, Mcl-1, BAR, BI-1 and Bcl-w (5). The pro-apoptotic family members include Bax, Bcl-xS, Bad, Bak, NBK, BID, Hrk, Bok, Bim, Noxa and Diva. Bax and Bak have been shown to play a critical role in cytochrome c release from mitochondria and thus initiate apoptosis (6). Bad plays a critical role in the Bax-mediated apoptosis pathway by dimerizing with Bcl-xL, causing the displacement of Bax. The displacement of Bax allows apoptosis to proceed (7). Bcl-xS, a shorter version of Bcl-xL (lacking amino acids 126-188), apparently utilizes a different pathway than Bax to induce cell death. Some research suggests that Bcl-xS uses a novel mechanism for regulating caspase or it may use an alternate cell death effector pathway (8,9).

<b>Form:</b>	Liquid
<b>Buffer:</b>	20 mM Tris-HCl, pH 8.0 , 10 mg/ml BSA, 0.05% Sodium Azide
<b>Storage:</b>	10 µl aliquots at -20°C. Avoid repeated freezing and thawing

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