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#9942

Pro-Apoptosis Bcl-2 Family Antibody Sampler Kit

1 Kit (8 x 20 microliters)

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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Bad (D24A9) Rabbit mAb	9239	20 µl	23 kDa	Rabbit IgG
Phospho-Bad (Ser112) (40A9) Rabbit mAb	5284	20 µl	23 kDa	Rabbit IgG
Bax (D2E11) Rabbit mAb	5023	20 µl	20 kDa	Rabbit IgG
Bik Antibody	4592	20 µl	20 kDa	Rabbit
Bim (C34C5) Rabbit mAb	2933	20 µl	12, 15, 23 kDa	Rabbit IgG
BID Antibody	2002	20 µl	15, 22 kDa	Rabbit
Bak (D4E4) Rabbit mAb	12105	20 µl	25 kDa	Rabbit IgG
Puma (D30C10) Rabbit mAb	12450	20 µl	23 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The Pro-Apoptosis Bcl-2 Family Antibody Sampler Kit provides an economical means to examine several members of the Bcl-2 family and their activation status. The kit contains enough primary and secondary antibodies to perform two Western blot experiments per primary antibody.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

Background

The Bcl-2 family consists of a number of evolutionarily conserved proteins containing Bcl-2 homology domains (BH) that regulate apoptosis through control of mitochondrial membrane permeability and release of cytochrome c (1-3). Four BH domains have been identified (BH1-4) that mediate protein interactions. The family can be separated into three groups based upon function and sequence homology: pro-survival members include Bcl-2, Bcl-xL, Mcl-1, A1 and Bcl-w; pro-apoptotic proteins include Bax, Bak and Bok; and "BH3 only" proteins Bad, Bik, Bid, Puma, Bim, Bmf, Noxa and Hrk. Interactions between death-promoting and death-suppressing Bcl-2 family members has led to a rheostat model in which the ratio of pro-apoptotic and anti-apoptotic proteins controls cell fate (4). Thus, pro-survival members exert their behavior by binding to and antagonizing death-promoting members. In general, the "BH3-only members" can bind to and antagonize the pro-survival proteins leading to increased apoptosis (5). While some redundancy of this system likely exists, tissue specificity, transcriptional and post-translational regulation of many of these family members can account for distinct physiological roles.

Bad is a pro-apoptotic member of the Bcl-2 family that can displace Bax from binding to Bcl-2 and Bcl-xL, resulting in cell death (6,7). Survival factors such as IL-3 can inhibit the apoptotic activity of Bad by activating intracellular signaling pathways that result in the phosphorylation of Bad at Ser112 and Ser136 (7). Phosphorylation at these sites results in the binding of Bad to 14-3-3 proteins and the inhibition of Bad binding to Bcl-2 and Bcl-xL (7). Akt has been shown to promote cell survival via its ability to phosphorylate Bad at Ser136 (8,9). Ser112 has been shown to be the substrate *in vivo* and *in vitro* of p90RSK (10,11) and mitochondria-anchored PKA (12).

Background References

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