Revision 1

Store at -20C

#17229

Pro-Survival Bcl-2 Family Antibody Sampler Kit II

1 Kit (7 x 20 microliters)

Cell Signaling TECHNOLOGY®

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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Bcl-2 (D55G8) Rabbit mAb	4223	20 µl	26 kDa	Rabbit IgG
Phospho-Bcl-2 (Ser70) (5H2) Rabbit mAb	2827	20 µl	28 kDa	Rabbit IgG
Mcl-1 (D2W9E) Rabbit mAb	94296	20 µl	40 (human), 35 (rodent) kDa	Rabbit IgG
Phospho-Mcl-1 (Thr163) (D5M9D) Rabbit mAb	14765	20 µl	40 kDa	Rabbit IgG
Bcl-xL (54H6) Rabbit mAb	2764	20 µl	30 kDa	Rabbit IgG
A1/Bfl-1 (D1A1C) Rabbit mAb	14093	20 µl	18 kDa	Rabbit IgG
Bcl-w (31H4) Rabbit mAb	2724	20 µl	18 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-Survival Bcl-2 Family Antibody Sampler Kit II provides an economical means to examine several members of the Bcl-2 family. The kit contains enough primary antibody to perform two western blot experiments.

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.
Several phosphorylation sites have been identified within Bcl-2 including Thr56, Ser70, Thr74 and Ser87 (6). These phosphorylation sites may be targets of the ASK1/MKK7/JNK1 pathway, and phosphorylation of Bcl-2 may be a marker for mitotic events (7,8). Mutation of Bcl-2 at Thr56 or Ser87 inhibits its anti-apoptotic activity during glucocorticoid-induced apoptosis of T lymphocytes (9). Interleukin 3 and JNK-induced Bcl-2 phosphorylation at Ser70 may be required for its enhanced antiapoptotic functions (10). Mcl-1 is phosphorylated in response to treatment with phorbol ester, microtubule-damaging agents, oxidative stress, and cytokine withdrawal (11-14). Phosphorylation at Thr163, the conserved MAP kinase/ERK site located within the PEST region, slows Mcl-1 protein turnover (13) but may prime the GSK-3 mediated phosphorylation at Ser159 that leads to Mcl-1 destabilization (14).

Background References

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