SANTA CRUZ BIOTECHNOLOGY, INC.

Bad (K-14): sc-6541

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The Bcl-2 family of proteins is characterized by its ability to modulate cell death (apoptosis) under a broad range of physiologic conditions. Bcl-2 and several related proteins function to inhibit apoptosis, while other members of the Bcl-2 family, such as Bax and Bak, enhance cell death under various conditions. For instance, Bcl-x_L represses cell death, while its shorter form, Bcl-x_S, promotes apoptosis. A protein designated Bad exhibits homology to Bcl-2, limited to the BH1 and BH2 domains. Bad functions to dimerize with Bcl-x_L and with Bcl-2, but not with Bax, Bcl-x_S, Mcl-1, A1 or itself. In mammalian cells, Bad binds with greater affinity to Bcl-x_L than to Bcl-2, and reverses the death repressor activity of Bcl-x_L but not Bcl-2. Dimerization of Bad with Bcl-x_L results in displacement of Bax from Bcl-x_L:Bax complexes, thereby causing restoration of Bax-mediated apoptosis.

REFERENCES

1. Nunez, G., et al. 1990. Deregulated Bcl-2 gene expression selectively prolongs survival of growth factor-deprived hemopoietic cell lines. J. Immunol. 144: 3602-3610.
2. Hockenbery, D.M., et al. 1991. Bcl-2 protein is topographically restricted in tissues characterized by apoptotic cell death. Proc. Natl. Acad. Sci. USA 88: 6961-6965.
3. Oltvai, Z.N., et al. 1993. Bcl-2 heterodimerizes *in vivo* with a conserved homolog, bax, that accelerates programmed cell death. Cell 74: 609-619.
4. Yin, X.M., et al. 1994. BH1 and BH2 domains of Bcl-2 are required for inhibition of apoptosis and heterodimerization with Bax. Nature 369: 321-323.
5. Gottschalk, A.R., et al. 1994. Identification of immunosuppressant-induced apoptosis in a murine B cell line and its prevention by Bcl-x but not Bcl-2. Proc. Natl. Acad. Sci. USA 91: 7350-7354.
6. Chittendon, T., et al. 1995. Induction of apoptosis by the Bcl-2 homologue Bak. Nature 374: 733-736.

CHROMOSOMAL LOCATION

Genetic locus: BAD (human) mapping to 11q13.1; Bad (mouse) mapping to 19 A.

SOURCE

Bad (K-14) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of Bad of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-6541 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

Bad (K-14) is recommended for detection of Bad of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for Bad siRNA (h): sc-29778, Bad siRNA (m): sc-29779, Bad shRNA Plasmid (h): sc-29778-SH, Bad shRNA Plasmid (m): sc-29779-SH, Bad shRNA (h) Lentiviral Particles: sc-29778-V and Bad shRNA (m) Lentiviral Particles: sc-29779-V.

Molecular Weight of Bad: 25 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200 or NIH/3T3 whole cell lysate: sc-2210.

SELECT PRODUCT CITATIONS

1. Adachi, S., et al. 2001. Cyclin A/Cdk2 activation is involved in hypoxia-induced apoptosis in cardiomyocytes. Circ. Res. 88: 408-414.
2. Yang, S.E., et al. 2002. Down-modulation of Bcl-x_L, release of cytochrome c and sequential activation of caspases during honokiol-induced apoptosis in human squamous lung cancer CH27 cells. Biochem. Pharmacol. 63: 1641-1651.
3. Wang, Q.F., et al. 2002. Regulation of Bcl-2 family molecules and activation of caspase cascade involved in gypenosides-induced apoptosis in human hepatoma cells. Cancer Lett. 183: 169-178.
4. Cheng, C.C., et al. 2005. Molecular mechanisms of ginsenoside Rh2-mediated G_1 growth arrest and apoptosis in human lung adenocarcinoma A549 cells. Cancer Chemother. Pharmacol. 55: 531-540.
5. Su, Y.T., et al. 2005. Emodin induces apoptosis in human lung adenocarcinoma cells through a reactive oxygen species-dependent mitochondrial signaling pathway. Biochem. Pharmacol. 70: 229-241.
6. Kuo, C.Y, et al. 2008. Functional characterization of hepatitis B virus X protein based on the inhibition of tumorigenesis in nude mice injected with CCL13-HBx cells. Intervirology 51: 253-260.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.