Bad (FL): sc-4702



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-x₁ 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_I 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-x₁ than to Bcl-2, and reverses the death repressor activity of Bcl-x_I 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., London, L., Hockenbery, D., Alexander, M. and McKearn, J.P. 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., Zutter, M., Hickey, W., Nahm, M. and Korsmeyer, S.J. 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., Milliman, C.L. and Korsmeyer, S.J. 1993. Bcl-2 heterodimerizes in vivo with a conserved homolog, Bax, that accelerates programmed cell death. Cell 74: 609-619.
- 4. Yin, X.-M., Oltvai, Z.N. and Korsmeyer, S.J. 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., Boise, L.H., Thompson, C.B. and Quintans, J. 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. Chittenden, T., Harrington, E.A., O'Connor, R., Flemington, C., Lutz, R.J., Evan, G.I. and Guild, B.C. 1995. Induction of apoptosis by the Bcl-2 homologue Bak. Nature 374: 733-736.
- 7. Kiefer, M.C., Brauer, M.J., Powers, V.C., Wu, J.J., Umansky, S.R., Tomei, L.D. and Barr, P.J. 1995. Modulation of apoptosis by the widely distributed Bcl-2 homologue Bak. Nature 374: 736-739.
- 8. Yang, E., Zha, J., Jockel, J., Boise, L.H., Thompson, C.B. and Korsmeyer, S.J. 1995. Bad, a heterodimeric partner for Bcl-x_l and Bcl-2, displaces Bax and promotes cell death. Cell 80: 285-291.

CHROMOSOMAL LOCATION

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

SOURCE

Bad (FL) is expressed in E. coli as a 45 kDa tagged fusion protein corresponding to amino acids 1-168 representing full length Bad of human origin.

PRODUCT

Bad (FL) is purified from bacterial lysates (>98%) by glutathione agarose affinity chromatography; supplied as 50 µg purified protein in PBS containing 5 mM DTT and 50% glycerol.

APPLICATIONS

Bad (FL) is suitable as Western blotting control for sc-942, sc-6541, sc-6542, sc-7869 and sc-8044.

STORAGE

Store at -20° C; stable for one year from the date of shipment.

RESEARCH USE

For research use only, not for use in diagnostic procedures

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