



## Bad (1-168): sc-4335 WB

### 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<sub>L</sub> represses cell death, while its shorter form, Bcl-x<sub>S</sub>, 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<sub>L</sub> and with Bcl-2, but not with Bax, Bcl-x<sub>S</sub>, Mcl-1, A1 or itself. In mammalian cells, Bad binds with greater affinity to Bcl-x<sub>L</sub> than to Bcl-2, and reverses the death repressor activity of Bcl-x<sub>L</sub> but not Bcl-2. Dimerization of Bad with Bcl-x<sub>L</sub> results in displacement of Bax from Bcl-x<sub>L</sub>: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 homology, 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. Chittenden, T., et al. 1995. Induction of apoptosis by the Bcl-2 homologue Bak. *Nature* 374: 733-736.
7. Kiefer, M.C., et al. 1995. Modulation of apoptosis by the widely distributed Bcl-2 homologue Bak. *Nature* 374: 736-739.
8. Yang, E., et al. 1995. Bad, a heterodimeric partner for Bcl-x<sub>L</sub> 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 (1-168) is expressed in *E. coli* as a 45 kDa tagged fusion protein corresponding to amino acids 1-168 of Bad of human origin.

### PRODUCT

Bad (1-168) is purified from bacterial lysates (>98%) by column chromatography; supplied as 10 µg in 0.1 ml SDS-PAGE loading buffer.

### APPLICATIONS

Bad (1-168) is suitable as a Western blotting control for sc-942, sc-942-G, sc-6541, sc-6542, sc-7869, sc-7998, sc-7998-G, sc-7999, sc-7999-G, sc-8044, sc-12969, sc-12969-G, sc-24561, sc-24599, sc-24600, sc-101640, sc-101641, sc-133356, sc-166932 and sc-271963.

Molecular Weight of Bad: 25 kDa.

### SELECT PRODUCT CITATIONS

1. Martin, L.J., et al. 2003. Early events of target deprivation/axotomy-induced neuronal apoptosis *in vivo*: oxidative stress, DNA damage, p53 phosphorylation and subcellular redistribution of death proteins. *J. Neurochem.* 85: 234-247.

### STORAGE

Store at -20° C. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

### RESEARCH USE

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

### PROTOCOLS

See our web site at [www.scbt.com](http://www.scbt.com) or our catalog for detailed protocols and support products.