



DB003: Bad (A17)

Background:

Bcl-2 family of proteins is a key regulator of apoptosis that function to either inhibit or promote cell death. The over expression of members such as Bcl-2 and Bcl-xL inhibit the apoptotic process (1,2). The Bcl-2 family members are also characterized by dimerizing to further modulate apoptosis. Bag-1, for example, has been found to form a heterodimer with Bcl-2 resulting in the enhancement of the anti-apoptotic effect of Bcl-2 (3,4). Other anti-apoptotic Bcl-2 family members include A1, Bcl-x_y, Bcl-x_β, Mcl-1, BAR, BI-1 and Bcl-w (5). The pro-apoptotic family members include Bax, Bcl-x_S, Bad, Bak, NBK, BID, Hrk, Bok, Bim, Noxa and Diva. Bax and Bak have been shown to play a critical role in cytochrome c release from mitochondria and thus initiate apoptosis (6). Bad plays a critical role in the Bax-mediated apoptosis pathway by dimerizing with Bcl-xL, causing the displacement of Bax. The displacement of Bax allows apoptosis to proceed (7). Bcl-x_S, a shorter version of Bcl-xL (lacking amino acids 126-188), apparently utilizes a different pathway than Bax to induce cell death. Some research suggests that Bcl-x_S uses a novel mechanism for regulating caspase or it may use an alternate cell death effector pathway (8,9).

Origin:

Bad is provided as an affinity purified rabbit polyclonal antibody, raised against a peptide mapping to the amino terminus of mouse Bad.

Product Details:

Each vial contains 200 µg/ml of affinity purified rabbit IgG, Bad *DB003 (A17)*, in 1 ml PBS containing 0.1 % sodium azide and 0.2% gelatin.

Competition Studies:

A blocking peptide is also available, *DB003P*, for use in competition studies. Each vial contains 100 µg of peptide in 0.5 ml PBS with 0.1% sodium azide and 100 µg BSA.

Specificity:

Bad *DB003(A17)* reacts with Bad of mouse and rat origin by western blotting and immunohistochemistry.

Storage:

Store this product at 4° C, do not freeze. The product is stable for one year from the date of shipment.

References:

1. Huang Z. 2000. Bcl-2 family proteins as targets for anticancer drug design. *Oncogene* 19(56): 6627-6631
2. Reed JC. 1997. Double identity for proteins of the Bcl-2 family. *Nature* 387(6635): 773-776
3. Eversole-Cire P, Concepcion FA, Simon MI, Takayama S, Reed JC, Chen J. 2000. Synergistic effect of Bcl-2 and BAG-1 on the prevention of photoreceptor cell death. *Invest Ophthalmol Vis Sci* 41(7): 1953-1961
4. Coldwell MJ, deSchoolmeester ML, Fraser GA, Pickering BM, Packham G, Willis AE. 2001. The p36 isoform of BAG-1 is translated by internal ribosome entry following heat shock. *Oncogene* 20(30): 4095-4100
5. Bae j, Hsu SY, Leo CP, Zell K, Hsueh AJ. 2001. Underphosphorylated BAD interacts with diverse antiapoptotic Bcl-2 family proteins to regulate apoptosis. *Apoptosis* 6(5): 319-330
6. Wei MC, Zong WX, Cheng EH, Lindsten T, Panoutsakopoulou V, Ross AJ, Roth KA, MacGregor GR, Thompson CB, Korsmeyer SJ. 2001. Proapoptotic BAX and BAK: a requisite gateway to mitochondrial dysfunction and death. *Science* 292(5517): 624-626
7. Yang E, Zha J, Jockel J, Boise LH, Thompson CB, Korsmeyer SJ. 1995. Bad, a heterodimeric partner for Bcl-XL and Bcl-2, displaces Bax and promotes cell death. *Cell* 80(2): 285-291
8. Fridman JS, Parsels J, Rehemtulla A, Maybaum J. 2001. Cytochrome c depletion upon expression of Bcl-XS. *J Biol Chem* 276(6): 4205-10
9. Lindenboim L, Yuan J, Stein R. 2000. Bcl-xS and Bax induce different apoptotic pathways in PC12 cells. *Oncogene* 19(14): 1783-1793