SANTA CRUZ BIOTECHNOLOGY, INC.

# Bad (N-19): sc-6542


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.

## CHROMOSOMAL LOCATION

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

## SOURCE

Bad (N-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-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-6542 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

## APPLICATIONS

Bad (N-19) 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), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Bad (N-19) is also recommended for detection of Bad in additional species, including canine.

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: Bad (h3): 293T Lysate: sc-170552, HeLa whole cell lysate: sc-2200 or NIH/3T3 + serum-starved cell lysate: sc-2257.

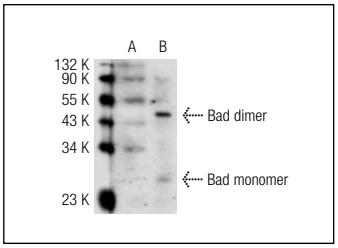
## STORAGE

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

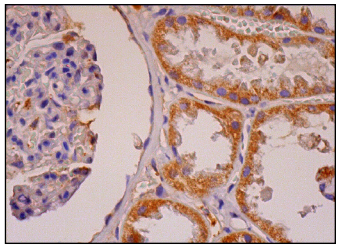
## RESEARCH USE

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

## DATA




Bad (N-19): sc-6542. Western blot analysis of Bad expression in non-transfected: sc-117752 (**A**) and human Bad transfected: sc-170552 (**B**) 293T whole cell lysates.



Bad (N-19): sc-6542. Immunoperoxidase staining of formalin fixed, paraffin-embedded human kidney tissue showing cytoplasmic staining of cells in tubules.

## SELECT PRODUCT CITATIONS

1. Royuela, M., et al. 2001. Immunoexpressions of p21, Rb, Mcl-1 and Bad gene products in normal, hyperplastic and carcinomatous human prostates. Eur. Cytokine Netw. 12: 654-663.
2. Kim, B.C., et al. 2002. Transforming growth factor β 1 induces apoptosis through cleavage of Bad in a Smad3-dependent mechanism in FaO hepatoma cells. Mol. Cell. Biol. 22: 1369-1378.
3. Claessens, Y.E., et al. 2002. *In vitro* proliferation and differentiation of erythroid progenitors from patients with myelodysplastic syndromes: evidence for FAS-dependent apoptosis. Blood 99: 1594-1601.
4. Marhaba, R., et al. 2003. CD44v7 interferes with activation-induced cell death by up-regulation of anti-apoptotic gene expression. J. Leukoc. Biol. 74: 135-148.
5. Bhakar, A.L., et al. 2003. Apoptosis induced by p75NTR overexpression requires Jun kinase-dependent phosphorylation of Bad. J. Neurosci. 23: 11373-11381.
6. Ramos, M., et al. 2011. Deficiency of the mitochondrial transporter of aspartate/glutamate aralar/AGC1 causes hypomyelination and neuronal defects unrelated to myelin deficits in mouse brain. J. Neurosci. Res. 89: 2008-2017.

## PROTOCOLS

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