

Product Information

BAD, GST-tagged, human recombinant, expressed in Sf9 insect cells

Catalog Number **SRP5164**
Storage Temperature -70°C

Synonyms: BBC2, BCL2L8

Product Description

BAD is a member of the BCL-2 family of proteins that are known to be regulators of programmed cell death. BAD is a pro-apoptotic protein that forms a heterodimer complex with BCL-xL and BCL-2, which reverses the prosurvival activity of these proteins.¹ The proapoptotic activity of BAD is regulated through its phosphorylation and this inhibits the pro-apoptosis function of BAD. Protein kinases such as AKT, RAF, and RSK1 can phosphorylate BAD and RSK1-induced phosphorylation of BAD at Ser¹¹² suppresses BAD-mediated apoptosis in neurons. BAD inhibits G₁ to S phase transition in MCF7 breast cancer cells and overexpression of BAD inhibits cell growth as well as cyclin D1 expression.²

Recombinant full-length human BAD was expressed by baculovirus in Sf9 insect cells using an N-terminal GST tag. The gene accession number is NM_032989. Recombinant protein stored in 50 mM Tris-HCl, pH 7.5, 150 mM NaCl, 10 mM glutathione, 0.1 mM EDTA, 0.25 mM DTT, 0.1 mM PMSF, and 25% glycerol.

Molecular mass: ~47 kDa

Purity: 70–95% (SDS-PAGE, see Figure 1)

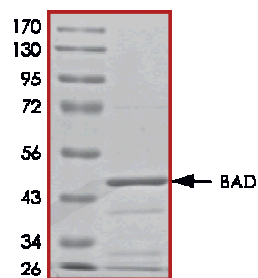
Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

The product ships on dry ice and storage at -70°C is recommended. After opening, aliquot into smaller quantities and store at -70°C . Avoid repeated handling and multiple freeze/thaw cycles.

Figure 1.
SDS-PAGE Gel of Typical Lot
70–95% (densitometry)



References

1. Yang, E. et al., Bad, a heterodimeric partner for Bcl-X(L) and Bcl-2, displaces Bax and promotes cell death. *Cell*, **80**, 285-291 (1995).
2. Fernando, R. et al., Breast cancer cell proliferation is inhibited by BAD: regulation of cyclin D1. *J. Biol. Chem.*, **282**(39), 28864-73 (2007).

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