

Phospho-Bad(S75) Antibody
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP3038a

Specification

Phospho-Bad(S75) Antibody - Product Information

Application	WB, IHC-P,E
Primary Accession	Q92934
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit Ig

Phospho-Bad(S75) Antibody - Additional Information

Gene ID 572

Other Names

Bcl2-associated agonist of cell death, BAD, Bcl-2-binding component 6, Bcl-2-like protein 8, Bcl2-L-8, Bcl-xL/Bcl-2-associated death promoter, Bcl2 antagonist of cell death, BAD, BBC6, BCL2L8

Target/Specificity

This Bad Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding S75 of human Bad.

Dilution

WB~~1:1000
IHC-P~~1:50~100

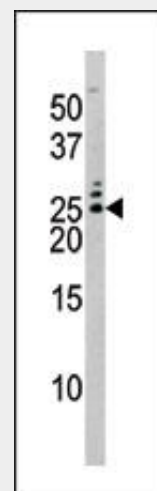
Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

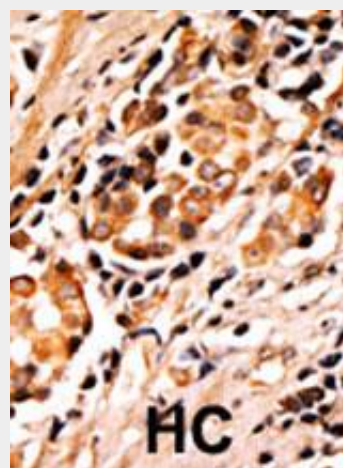
Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions



The anti-Phospho-Bad-S75 Pab (Cat. #AP3038a) is used in Western blot to detect Phospho-Bad-S75 in HL60 tissue lysate



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

Phospho-Bad(S75) Antibody - Background

Phospho-Bad(S75) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Phospho-Bad(S75) Antibody - Protein Information

Name BAD

Synonyms BBC6, BCL2L8

Function

Promotes cell death. Successfully competes for the binding to Bcl-X(L), Bcl-2 and Bcl-W, thereby affecting the level of heterodimerization of these proteins with BAX. Can reverse the death repressor activity of Bcl-X(L), but not that of Bcl-2 (By similarity). Appears to act as a link between growth factor receptor signaling and the apoptotic pathways.

Cellular Location

Mitochondrion outer membrane. Cytoplasm {ECO:0000250|UniProtKB:Q61337}.
Note=Colocalizes with HIF3A in the cytoplasm (By similarity). Upon phosphorylation, locates to the cytoplasm. {ECO:0000250|UniProtKB:Q61337}

Tissue Location

Expressed in a wide variety of tissues.

Phospho-Bad(S75) Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Bad is a member of the BCL-2 family. BCL-2 family members are known to be regulators of programmed cell death. This protein positively regulates cell apoptosis by forming heterodimers with BCL-xL and BCL-2, and reversing their death repressor activity. Proapoptotic activity of this protein is regulated through its phosphorylation. Protein kinases AKT and MAP kinase, as well as protein phosphatase calcineurin are found to be involved in the regulation of this protein. Bad is phosphorylated on one or more of Ser-75, Ser-99, Ser-118 and Ser-134 in response to survival stimuli, which blocks its pro-apoptotic activity. Phosphorylation on Ser-99 or Ser-75 promotes heterodimerization with 14-3-3 proteins. This interaction then facilitates the phosphorylation at Ser-118, a site within the BH3 motif, leading to the release of Bcl-X(L) and the promotion of cell survival. Ser-99 is the major site of AKT/PKB phosphorylation, Ser-118 the major site of protein kinase A (CAPK) phosphorylation

Phospho-Bad(S75) Antibody - References

Hurbin, A., et al., J. Biol. Chem. 280(20):19757-19767 (2005).
Antignani, A., et al., Biochemistry 44(10):4074-4082 (2005).
Ying, S., et al., Infect. Immun. 73(3):1399-1403 (2005).
Seo, S.Y., et al., J. Biol. Chem. 279(40):42240-42249 (2004).
Lee, J.W., et al., Carcinogenesis 25(8):1371-1376 (2004).