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Anti-Bad (Ser-112), Phosphospecific Antibody

Catalog # AN1653

Product Information

Application	WB
Primary Accession	Q61337
Host	Rabbit
Clonality	Rabbit Polyclonal
Isotype	IgG
Calculated MW	22080

Additional Information

Gene ID	12015
Other Names	Bcl2 antagonist of cell death, BAD; Bcl-2-binding component 6, Bbc6, Bcl-xL/Bcl-2-associated death promoter
Target/Specificity	Bad is a member of the BCL-2 family of regulators involved in 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 IKK, and MAP kinases, as well as protein phosphatase calcineurin are found to be involved in the regulation of this Bad activity. Phosphorylation of Bad occurs on one or more of Ser-26, Ser-112, Ser-136, and Ser-155 in response to survival stimuli, which blocks its pro-apoptotic activity. Phosphorylation on Ser-136 or Ser-112 promotes heterodimerization with 14-3-3 proteins. This interaction then facilitates the phosphorylation at Ser-155, a site within the BH3 motif, leading to the release of Bcl-xL and the promotion of cell survival. Ser-26 is phosphorylated by IKK leading to phosphorylation of C-terminal serine sites and disruption of binding to Bcl-xL. This inactivation of Bad inhibits TNFα-induced apoptosis independent of NF-κB activity.
Dilution	WB~~1:1000
Format	Antigen Affinity Purified
Storage	Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	Anti-Bad (Ser-112), Phosphospecific Antibody is for research use only and not for use in diagnostic or therapeutic procedures.
Shipping	Blue Ice

Background

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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 IKK, and MAP kinases, as well as protein phosphatase calcineurin are found to be involved in the regulation of this Bad activity. Phosphorylation of Bad occurs on one or more of Ser-26, Ser-112, Ser-136, and Ser-155 in response to survival stimuli, which blocks its pro-apoptotic activity. Phosphorylation on Ser-136 or Ser-112 promotes heterodimerization with 14-3-3 proteins. This interaction then facilitates the phosphorylation at Ser-155, a site within the BH3 motif, leading to the release of Bcl-xL and the promotion of cell survival. Ser-26 is phosphorylated by IKK leading to phosphorylation of C-terminal serine sites and disruption of binding to Bcl-xL. This inactivation of Bad inhibits TNFα-induced apoptosis independent of NF-κB activity.