

# Anti-Bad (Ser-26), Phosphospecific Antibody

Catalog # AN1652

## Product Information

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<b>Application</b>	WB
<b>Primary Accession</b>	<a href="#">Q61337</a>
<b>Host</b>	Rabbit
<b>Clonality</b>	Rabbit Polyclonal
<b>Isotype</b>	IgG
<b>Calculated MW</b>	22080

## Additional Information

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<b>Gene ID</b>	12015
<b>Other Names</b>	Bcl2 antagonist of cell death, BAD; Bcl-2-binding component 6, Bbc6, Bcl-xL/Bcl-2-associated death promoter
<b>Target/Specificity</b>	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 TNF $\alpha$ -induced apoptosis independent of NF- $\kappa$ B activity.
<b>Dilution</b>	WB~~1:1000
<b>Format</b>	Antigen Affinity Purified
<b>Storage</b>	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.
<b>Precautions</b>	Anti-Bad (Ser-26), Phosphospecific Antibody is for research use only and not for use in diagnostic or therapeutic procedures.
<b>Shipping</b>	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 TNF $\alpha$ -induced apoptosis independent of NF- $\kappa$ B activity.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.