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[Marker Primary Antibody]

Rabbit Anti-Phospho-Bad (Ser118)/HRP Conjugated antibody

Catalog Number: SL5216R-HRP

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	Anti-Phospho-Bad (Ser118)/HRP
Immunogen:	KLH conjugated Synthesised phosphopeptide derived from human BAD around the phosphorylation site of Ser118
Purification:	affinity purified by Protein G
Concentration:	1mg/ml
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Applications:	WB=1:500-2000 IHC-P=1:100-500 IHC-F=1:100-500 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	Human,Mouse,Rat(predicted:Dog,Pig,Cow,Horse,Rabbit)
Background:	background: Bad is a member of the Bcl2 family and acts to promote apoptosis by forming heterodimers with the survival proteins Bcl2 and BclxL, thus preventing them from binding with BAX. Bad is found on the outer mitochondrial membrane and, once phosphorylated in response to growth stimuli, translocates to the cytoplasm. The phosphorylation status of Bad represents a key checkpoint for death or cell survival. JNK-induced phosphorylation of BAD serine 128 promotes the apoptotic role of Bad by opposing the inhibitory effect of growth factor on Bad-mediated apoptosis. Cdc2-induced phosphorylation of Bad serine 128 has an inhibitory effect on its interaction with 14-3-3 proteins. The latter interaction is critical for Bad phosphorylation at serine 155, a site within the BH3 domain that leads to the release of BclxL and the promotion of cell survival.

Important Note: This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

	Alternative splicing of this gene results in two transcript variants which encode the same isoform. **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. Appears to act as a link between growth factor receptor signaling and the apoptotic pathways. **Subunit:** Forms heterodimers with the anti-apoptotic proteins, Bcl-X(L), Bcl-2 and Bcl-W. Also binds protein S100A10. The Ser-75/Ser-99 phosphorylated form binds 14-3-3 proteins. Interacts with AKT1 and PIM3. **Subcellular Location:** Mitochondrion outer membrane. Cytoplasm. Upon phosphorylation, locates to the cytoplasm. **Tissue Specificity:** Expressed in a wide variety of tissues. **Post-translational modifications:** Phosphorylated on one or more of Ser-75, Ser-99, Ser-118 andSer-134 in response to survival stimuli, which blocks itspro-apoptotic activity. Phosphorylation on Ser-99 or Ser-75promotes heterodimerization with 14-3-3 proteins. This interactionthen facilitates the phosphorylation at Ser-118, a site within theBH3 motif, leading to the release of Bcl-X(L) and the promotion ofcell survival. Ser-99 is the major site of AKT/PKB phosphorylation, Ser-118 the major site of protein kinase A (CAPK) phosphorylation. Phosphorylation at Ser-99 by PKB/AKT1 is almost completely blockedby the apoptotic C-terminus cleavage product of PKN2 generated bycaspases-3 activity during apoptosis. Methylation at Arg-94 and Arg-96 by PRMT1 inhibits Akt-mediated phosphorylation at Ser-99. **Similarity:** Belongs to the Bcl-2 family. **Database links:** **Entrez Gene:** 572 Human **Entrez Gene:** 12015 Mouse **Entrez Gene:** 64639 Rat Omim: 603167 Human **SwissProt:** Q92934 Human **SwissProt:** Q61337 Mouse **SwissProt:** O35147 Rat **Unigene:** 370254 Human **Unigene:** 4387 Mouse **Unigene:** 36696 Rat Important **Note:** This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

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	BAD is a negative regulator gene of BCL2/BAX and BCL-XL/BAX heterodimers. BAD is a BCL2/BCL-XL-associated death promoter, acting as a BCL2 and BCL-XL heterodimer partner to promote apoptosis.

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