

## Basic Information

<b>Product Name</b>	Anti-BAD (Phospho-S112) Antibody
<b>Gene Name</b>	BAD
<b>Source</b>	Rabbit
<b>Isotype</b>	IgG
<b>Species Reactivity</b>	human, mouse, rat
<b>Tested Application</b>	WB
<b>Contents</b>	500 ug/ml; Rabbit IgG in phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.
<b>Immunogen</b>	A synthesized peptide derived from human Bad 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.
<b>concentration</b>	500 ug/ml
<b>Purification</b>	Affinity-chromatography
<b>Observed MW</b>	23KD
<b>Dilution Ratios</b>	Western blot (WB):1:500-2000

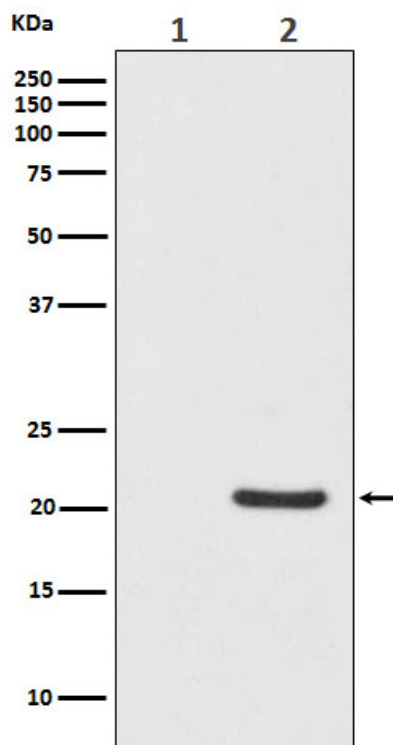
## Storage

12 months from date of receipt, -20°C as supplied. 6 months 2 to 8°C after reconstitution. Avoid repeated freezing and thawing.

## Background Information

Bad is a proapoptotic member of the Bcl-2 family that promotes cell death by displacing Bax from binding to Bcl-2 and Bcl-xL (1,2). Survival factors, such as IL-3, inhibit the apoptotic activity of Bad by activating intracellular signaling pathways that result in the phosphorylation of Bad at Ser112 and Ser136 (2). Phosphorylation at these sites promotes binding of Bad to 14-3-3 proteins to prevent an association between Bad with Bcl-2 and Bcl-xL (2). Akt phosphorylates Bad at Ser136 to promote cell survival (3,4). Bad is phosphorylated at Ser112 both in vivo and in vitro by p90RSK (5,6) and mitochondria-anchored PKA (7). Phosphorylation at Ser155 in the BH3 domain by PKA plays a critical role in blocking the dimerization of Bad and Bcl-xL (8-10).

## Selected Validation Data



Western blot analysis of Phospho-Bad (S112) expression in (1) HeLa cell lysate; (2) HeLa cell treated with Calcyculin A lysate.