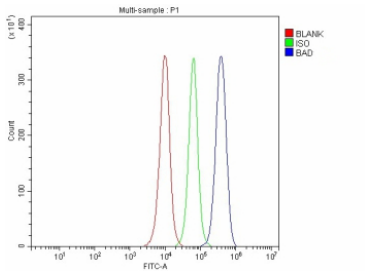
BAD Antibody / Bcl2-associated agonist of cell death (FY13327)

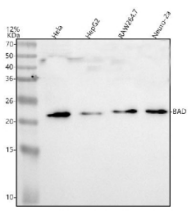
Catalog No.	Formulation	Size
FY13327	Adding 0.2 ml of distilled water will yield a concentration of 500 ug/ml	100 ug

Bulk quote request

Availability	1-2 days
Species Reactivity	Human, Mouse
Format	Lyophilized
Clonality	Polyclonal (rabbit origin)
Isotype	Rabbit IgG
Purity	Immunogen affinity purified
Buffer	Each vial contains 4 mg Trehalose, 0.9 mg NaCl, 0.2 mg Na2HPO4.
UniProt	Q92934
Applications	Western Blot : 0.25-0.5ug/ml
Limitations	This BAD antibody is available for research use only.



Flow Cytometry analysis of human JK cells using anti-BAD antibody. Overlay histogram showing JK cells stained with (Blue line). To facilitate intracellular staining, cells were fixed with 4% paraformaldehyde and permeabilized with permeabilization buffer. The cells were blocked with 10% normal goat serum. And then incubated with rabbit anti-BAD antibody (1 ug/million cells) for 30 min at 20oC. DyLight 488 conjugated goat anti-rabbit IgG (5-10 ug/million cells) was used as secondary antibody for 30 minutes at 20oC. Isotype control antibody (Green line) was rabbit IgG (1 ug/million cells) used under the same conditions. Unlabelled sample without incubation with primary antibody and secondary antibody (Red line) was used as a blank control.



Western blot analysis of BAD using anti-BAD antibody. Lane 1: human Hela whole cell lysates, Lane 2: human HepG2 whole cell lysates, Lane 3: mouse Raw264.7 whole cell lysates, Lane 4: mouse Neuro-2a whole cell lysates. After electrophoresis, proteins were transferred to a nitrocellulose membrane at 150 mA for 50-90 minutes. Blocked the membrane with 5% non-fat milk/TBS for 1.5 hour at RT. The membrane was incubated with rabbit anti-BAD antibody at 0.5 ug/ml overnight at 4oC, then washed with TBS-0.1%Tween 3 times with 5 minutes each and probed with a goat anti-rabbit IgG-HRP secondary antibody at a dilution of 1:5000 for 1.5 hour at RT. The signal was developed using enhanced chemiluminescent. The commonly observed molecular weight of BAD is 19-23 kDa.

Description

BAD antibody detects Bcl2-associated agonist of cell death, a pro-apoptotic member of the BCL2 protein family encoded by the BAD gene on chromosome 11q13.1. BAD is a cytoplasmic and mitochondrial-associated protein that regulates apoptosis by controlling mitochondrial outer membrane permeability. It is highly expressed in lymphoid tissues, liver, heart, and neurons, where it serves as a pivotal switch between cell survival and programmed cell death. BAD promotes apoptosis by binding to anti-apoptotic proteins BCL2 and BCL-XL, thereby freeing pro-apoptotic effectors such as BAX and BAK to induce cytochrome c release and caspase activation.

Under normal growth conditions, BAD remains phosphorylated and inactive, sequestered in the cytosol through association with 14-3-3 scaffold proteins. Upon cellular stress or growth factor deprivation, BAD becomes dephosphorylated by phosphatases such as PP2A, leading to its translocation to mitochondria and initiation of apoptosis. Functionally, BAD integrates signals from multiple pathways, including PI3K-AKT, MAPK, and cAMP-PKA, which regulate its phosphorylation state and apoptotic potential. Thus, BAD acts as a key convergence point for survival and death signals in diverse cell types.

Structurally, BAD contains BH3 (Bcl2 homology 3) domains essential for heterodimerization with other BCL2 family proteins. This domain mediates selective binding that determines the balance between apoptosis inhibition and activation. Post-translational modifications such as phosphorylation at serine residues (Ser112, Ser136, and Ser155) and dephosphorylation events fine-tune BAD activity in response to extracellular cues. The protein localizes to mitochondria during apoptosis induction, where it promotes permeabilization of the mitochondrial outer membrane and triggers the intrinsic apoptotic cascade.

Dysregulation of BAD expression or phosphorylation contributes to cancer, neurodegenerative diseases, and ischemic injury. Overactivation of BAD leads to neuronal loss in neurodegenerative conditions, while reduced activity supports tumor cell survival and resistance to therapy. In cancer research, BAD phosphorylation status serves as a marker for apoptosis sensitivity and therapeutic response. BAD also plays a metabolic role in glucose homeostasis, influencing insulin secretion and mitochondrial respiration in pancreatic beta cells.

BAD is a member of the BCL2 protein family, which governs mitochondrial apoptotic pathways by regulating membrane permeability and caspase activation. Pathway associations include the intrinsic apoptosis pathway and PI3K-AKT signaling, which modulate BAD phosphorylation and cellular survival. The protein's regulatory versatility makes it an important target in oncology, neurology, and metabolic research.

Immunohistochemical staining using BAD antibody reveals cytoplasmic and mitochondrial localization in apoptotic cells. The BAD antibody from NSJ Bioreagents is a reliable tool for studying apoptosis regulation, mitochondrial signaling, and stress-induced cell death mechanisms.

Application Notes

Optimal dilution of the BAD antibody should be determined by the researcher.

Immunogen

A synthetic peptide corresponding to a sequence in the middle region of human BAD was used as the immunogen for the BAD antibody.

Storage

After reconstitution, the BAD antibody can be stored for up to one month at 4oC. For long-term, aliquot and store at -20oC. Avoid repeated freezing and thawing.