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BID Polyclonal Antibody

Catalog Number **PA1-22010**

Product data sheet

Details	
Size	50 µL
Host/Isotope	Rabbit / IgG
Class	Polyclonal
Type	Antibody
Immunogen	Synthetic peptide corresponding to a region of human BID.
Conjugate	Unconjugated
Form	Liquid
Concentration	0.5 mg/mL
Storage Conditions	Store at 4°C short term. For long term storage, store at -20°C, avoiding freeze/thaw cycles.

Species Reactivity	
Species reactivity	Human

Tested Applications	Dilution *
Western Blot (WB)	0.25-1 µg/mL

* Suggested working dilutions are given as a guide only. It is recommended that the user titrate the product for use in their own experiment using appropriate negative and positive controls.

Product specific information

PA1-22010 detects Bid Cleavage Site in human samples. This antibody specifically detects the ~15 kDa, C-terminal cleavage fragment of human BID (p15). PA1-22010 has been successfully used in Western blot procedures. Positive control of Jurkat cells treated with anti Fas antibody to induce cleavage suggested. The PA1-22010 immunogen is a synthetic peptide corresponding to a region of human BID. Store at 4°C short term. For extended storage aliquot and store at -20°C or below. Avoid freeze/thaw cycles.

Background/Target Information

BID is a death agonist that heterodimerizes with either agonist BAX or antagonist BCL2. Bid is a member of the BCL-2 family of cell death regulators. BID is a mediator of mitochondrial damage induced by caspase-8 (CASP8); CASP8 cleaves this encoded protein, and the COOH-terminal part translocates to mitochondria where it triggers cytochrome c release. BID is also a pro-apoptotic member of the Bcl 2 family. BID usually exists in an inactive form in the cytosolic fraction of living cells and becomes cleaved and activated by caspase 8 in response to TNF alpha or Fas ligand. Once BID is cleaved, the C-terminal 15 kDa fragment of BID (p15) translocates onto mitochondria and is sufficient to trigger cytochrome c release, resulting in cell apoptosis. BID serves as a direct molecular link between caspase 8 activation and mitochondrial death machinery.

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