Human/Mouse BID Alexa Fluor® 700-conjugated Antibody

Antigen Affinity-purified Polyclonal Goat IgG
Catalog Number: AF860N
100 µg

DESCRIPTION

Species Reactivity	Human/Mouse
Specificity	Detects human and mouse BID, a fragment generated in cells treated with anti-Fas and the carboxyl-terminal portion of recombinant BID generated by cleavage with Caspase 8 in Western blots.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	*E. coli*-derived recombinant mouse BID Met1-Asp195 Accession # AAC71064
Conjugate	Alexa Fluor 700 Excitation Wavelength: 675-700 nm Emission Wavelength: 723 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunoprecipitation	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

BID is a 195 amino acid member of the Bcl-2 family of proteins that regulates outer mitochondrial membrane permeability (1). BID is a pro-apoptotic member that causes cytochrome c to be released from the mitochondria intermembrane space into the cytosol. In healthy cells BID is cytosolic. In response to Fas ligand or TNF, BID is cleaved by caspase-8 and it then relocates to the mitochondria outer membrane (2, 3). Cleavage of BID by caspase-8 generates a new N-terminal that contains a terminal glycine. It appears that the glycine is myristoylated and myristoylation serves to target BID to the mitochondria (4). BID may then interact with another pro-apoptotic Bcl-2 family member Bak (5). Interaction of BID with Bak causes altered mitochondrial membrane permeability. A 9-13 amino acid stretch called the BH3 region (Bcl-2 homology region) appears to mediate the BID interaction with other Bcl-2 family members. BID is neutralized by binding to the anti-apoptotic member Bcl-xL.

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