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Bid Antibody (N-term S65) Blocking peptide
Synthetic peptide
Catalog # BP12412a

Specification

Bid Antibody (N-term S65) Blocking peptide - Product Information

Primary Accession P55957

Bid Antibody (N-term S65) Blocking peptide - Additional Information

Gene ID 637

Other Names
BH3-interacting domain death agonist, p22 BID, BID, BH3-interacting domain death agonist p15, p15 BID, BH3-interacting domain death agonist p13, p13 BID, BH3-interacting domain death agonist p11, p11 BID, BID

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Bid Antibody (N-term S65) Blocking peptide - Protein Information

Name BID

Function
Induces caspases and apoptosis (PubMed:14583606). Counters the protective effect of BCL2 (By similarity).

Cellular Location
Cytoplasm. Mitochondrion membrane. Mitochondrion outer membrane. Note=When uncleaved, it is predominantly cytoplasmic. [BH3-interacting domain death agonist p13]: Mitochondrion membrane {ECO:0000250|UniProtKB:P70444}. Note=Associated with the mitochondrial membrane. {ECO:0000250|UniProtKB:P70444} [Isoform 3]: Cytoplasm

Tissue Location
[Isoform 2]: Expressed in spleen, pancreas and placenta (at protein level). [Isoform 4]: Expressed in lung and pancreas (at protein level).

Bid Antibody (N-term S65) Blocking peptide - Protocols

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Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

Bid Antibody (N-term S65) Blocking peptide - Images

Bid Antibody (N-term S65) Blocking peptide - Background

Bid, a BH3 domain containing proapoptotic Bcl2 family member, is localized in the cytosolic fraction of cells as an inactive precursor. Its active form is generated upon proteolytic cleavage by caspase 8 in the Fas signaling pathway. Cleaved Bid translocates to mitochondria and releases its potent proapoptotic activity, which in turn induces cytochrome c release and mitochondrial damage. The cytochrome c releasing activity of Bid was antagonized by Bcl2. Mutation in the SH3 domain can diminish the cytochrome c releasing activity. In animal model studies, Bid deficient mice are found resistant to the lethal effects of death factor signals relayed through Fas.