Recombinant Protein Technical Manual

Recombinant Human BID Protein (Active)

RPES2923

Product Data:

Product SKU: RPES2923

Size: 50µg

Species: Human

Expression host: E. coli

Uniprot: P55957

Protein Information:

Molecular Mass:

AP Molecular Mass: 22 kDa

Tag:

Bio-activity: 1. Measured by its binding ability in a functional ELISA.2. Immobilized human BID at 10 µg/mL (100 µl/well) can bind biotinylated human BCL2L1, The EC50 of biotinylated human BCL2L1 is 7.1 ng/mL.3. Immobilized human BID at 10 µg/mL (100 µl/well) can bind biotinylated mouse BCL2L1, The EC50 of biotinylated mouse BCL2L1 is 5.6 ng/mL.

Purity: > 90 % as determined by reducing SDS-PAGE.

Endotoxin: Please contact us for more information.

Storage: Lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.

Shipping: This product is provided as lyophilized powder which is shipped with ice packs.

Formulation: Lyophilized from sterile 40mM Tris, 150mM NaCl, pH 8.0

Reconstitution: Please refer to the printed manual for detailed information.

Application: Functional ELISA

Synonyms: BH3-Interacting Domain Death Agonist; p22 BID; BID

Immunogen Information:

Sequence: Met 1-Asp 195

Background:

The BH3 interacting domain death agonist (BID) is a pro-apoptotic member of the Bcl-2 protein family, which contains only the BH3 domain, and is required for its interaction with the Bcl-2 family proteins and for its pro-death activity. BID is important to cell death mediated by these proteases and thus is the sentinel to protease-mediated death signals. Recent studies further indicate that Bid may be more than just a killer molecule, it could be also involved in the maintenance of genomic stability by engaging at mitosis checkpoint. BID is an integrating key regulator of the intrinsic death pathway that amplifies caspase-dependent and caspase-independent execution of neuronal apoptosis. Therefore pharmacological inhibition of BID provides a promising therapeutic strategy in neurological diseases where programmed cell death is prominent. BID is activated by Caspase 8 in response to Fas/TNF-R1 death receptor activation. Activated BID is translocated to mitochondria and induces cytochrome c release, which in turn activates downstream caspases. BID action has been proposed to involve the mitochondrial re-location of its truncated form, tBid, to facilitate the release of apoptogenic proteins like cytochrome c.