

Recombinant Human BID Protein

Catalog Number: PKSH031587

Note: Centrifuge before opening to ensure complete recovery of vial contents.

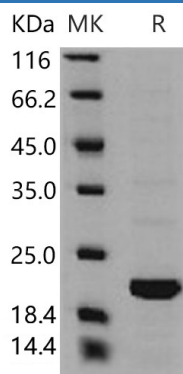
Description

Species	Human
Source	E.coli-derived Human BID protein Met 1-Asp 195
Calculated MW	22 kDa
Observed MW	22 kDa
Accession	P55957-1
Bio-activity	1. Immobilized human BID at 10 µg/mL (100 µl/well) can bind biotinylated human BCL2L1, The EC ₅₀ of biotinylated human BCL2L1 is 7.1 ng/mL. 2. Immobilized human BID at 10 µg/mL (100 µl/well) can bind biotinylated mouse BCL2L1, The EC ₅₀ of biotinylated mouse BCL2L1 is 5.6 ng/mL.

Properties

Purity	> 90 % as determined by reducing SDS-PAGE.
Endotoxin	Please contact us for more information.
Storage	Generally, 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°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C 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 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Reconstitution	Please refer to the specific buffer information in the printed manual. Please refer to the printed manual for detailed information.

Data



> 90 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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.