Recombinant Mouse BID Protein (His &GST Tag)

Catalog Number: PKSM040720

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

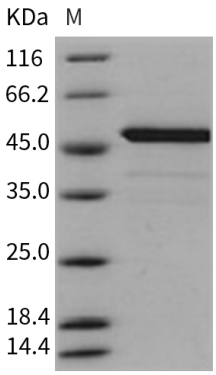
Description

Species	Mouse
Source	E.coli-derived Mouse BID protein Met 1-Asp 195, with an N-terminal His & GST
Calculated MW	50.0 kDa
Observed MW	47 kDa
Accession	EDK99650.1
Bio-activity	1. Immobilized mouse BID at 10 μg/mL (100 μl/well) can bind biotinylated human BCL2L1, The EC_{50} of biotinylated human BCL2L1 is 7.01 ng/mL. 2. Immobilized mouse BID at 10 μg/mL (100 μl/well) can bind biotinylated mouse BCL2L1, The EC_{50} of biotinylated mouse BCL2L1 is 7.1 ng/mL.

Properties

Purity	> 95 % 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 ℃. 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 50mM Tris, 150mM NaCl, pH 7.5 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 95 % as determined by reducing SDS-PAGE.

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.

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