

## Recombinant Human BID Protein

Catalog Number: PKSH031587

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

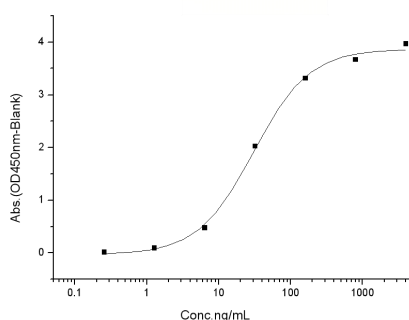
### Description

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

### Properties

<b>Purity</b>	> 90 % as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	Please contact us for more information.
<b>Storage</b>	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.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	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. Please refer to the specific buffer information in the printed manual.
<b>Reconstitution</b>	Please refer to the printed manual for detailed information.

### Data



Immobilized Human BID (Cat: PKSH031587) at 2 µg/mL (100 µL/well) can bind Human Bcl-XL His (Cat: PKSH031599), the EC<sub>50</sub> of Human Bcl-XL His (Cat: PKSH031599) is 15-80 ng/mL.

### 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.

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