

# Recombinant Mouse BID Protein (His & GST Tag)

Catalog Number:PKSM040720



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

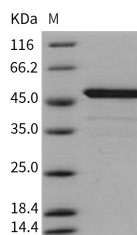
## Description

<b>Synonyms</b>	2700049M22Rik;AI875481;AU022477
<b>Species</b>	Mouse
<b>Expression Host</b>	E.coli
<b>Sequence</b>	Met 1-Asp 195
<b>Accession</b>	EDK99650.1
<b>Calculated Molecular Weight</b>	50.0 kDa
<b>Observed molecular weight</b>	47 kDa
<b>Tag</b>	N-His-GST
<b>Bioactivity</b>	1. Immobilized mouse BID at 10 µg/mL (100 µl/well) can bind biotinylated human BCL2L1, The EC50 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 EC50 of biotinylated mouse BCL2L1 is 7.1 ng/mL.

## Properties

<b>Purity</b>	> 95 % 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 50mM Tris, 150mM NaCl, pH 7.5 Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 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



> 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

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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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