Recombinant Human BID Protein

Catalog Number: PKSH033417

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

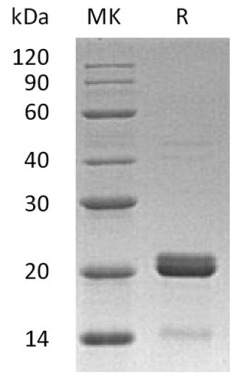
Description

Species	Human
Mol_Mass	22.0 kDa
Accession	P55957
Bio-activity	Not validated for activity

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < - 20°C.
Formulation	Supplied as a 0.2 μm filtered solution of 20mM PB, 100mM KCl, pH 7.4.
Reconstitution	Not Applicable

Data



> 95 % as determined by reducing SDS-PAGE.

Background

BH3-Interacting Domain Death Agonist (BID) is a member of the Bcl-2 protein family which regulates outer mitochondrial membrane permeability. BID is a pro-apoptotic member that causes cytochrome c to be released from the mitochondria intermembrane space into the cytosol. BID contains only the BH3 domain; which is required for its interaction with the Bcl-2 family proteins and for its pro-death activity. BID is susceptible to proteolytic cleavage by caspases; calpains; Granzyme B and cathepsins. It 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; and also offer a new strategy for the treatment of acute renal failure associated with ischemia-reperfusion. BID receives direct inputs from a key regulator of the cell cycle arrest/DNA repair machinery (ATM); and therefore is an excellent candidate to coordinate genotoxic stress responses and apoptotic cell death. BID is a novel pro-apoptosis Bcl-2 family protein that is activated by caspase 8 in response to Fas/TNF-R1 death receptor signals. Deletion of BID inhibits carcinogenesis in the liver; although this genetic alteration promotes tumorigenesis in the myeloid cells. This is likely related to the function of BID to promote cell cycle progression into S phase. BID could be also involved in the maintenance of genomic stability by engaging at mitosis checkpoint.

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