

Recombinant Human cIAP1/HiAP2 Protein (AVI Tag)

Catalog Number:PKSH031263



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

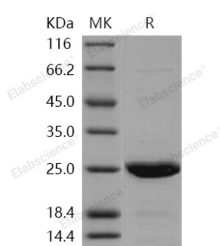
Description

Synonyms	API1;c-IAP1;cIAP1;Hiap-2;HIAP2;MIHB;RNF48
Species	Human
Expression Host	E.coli
Sequence	Glu 144-Leu 356
Accession	NP_001157.1
Calculated Molecular Weight	26.5 kDa
Observed molecular weight	26.5 kDa
Tag	C-Avi
Bioactivity	Measured by its ability to inhibit DEVD-AFC cleavage activity in cell extracts activated by addition of cytochrome c and dATP. The IC50 for this effect is typically 25-750 nM.

Properties

Purity	> 92 % 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 10mM Tris, 5% glycerol, 0.5mM EDTA, 5mM DTT, 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 ma
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 92 % as determined by reducing SDS-PAGE.

Background

The cellular inhibitor of apoptosis protein-1 (cIAP1) is a member of the Inhibitor of Apoptosis family proteins (IAP) whose members are characterized by a novel domain of about 70 amino acids termed baculoviral IAP repeats (BIRs). The BIR domains of cIAP1 and cIAP2 bind to caspases, the key effector proteases of apoptosis. The IAP protein family which can enhance cell survival are crucial regulators of programmed cell death. Both cIAP1 and cIAP2 are the E3

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ubiquitin protein isopeptide ligases for Smac, taking part in promoting cancer survival through functioning as E3 ubiquitin ligases. Removal of cIAP1 by genetic deletion may result in NF- κ B signaling activation that induces TNF α production and in killing sensitive tumor cells through enhanced TNF-R1 death-receptor signaling and caspase 8 activation. The substrate-dependent E3 activity of cIAPs is mediated by their RING domains and is dependent on the specific interactions between cIAPs and Smac. cIAP1 and cIAP2 are also reported to be regulators of NF- κ B activation upon TNF treatment.

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