Data Sheet (Cat.No.TMAY-00871)

TargetMol

Anti-BID Antibody (1W750)

Product Details

Ig Type:	Rabbit IgG
Reactivity:	Human
Conjugation:	Unconjugated
Clone:	1W750
Purification:	Protein A

Applications

Verified Activity:	Immunofluorescence staining of Human BID in Hela cells. Cells were fixed with 4% PFA, permeabilzed with 0.3% Triton X-100 in PBS, blocked with 10% serum, and incubated with rabbit anti-Human BID monoclonal antibody (1:60) at 37°C 1 hour. Then cells were stained with the Alexa Fluor® 488-conjugated Goat Anti-rabbit IgG secondary antibody (green) and counterstained with DAPI (blue). Positive staining was localized to cytoplasm.
Application:	ELISA,ICC/IF
Recommended	ELISA: 1:5000-1:10000; ICC-IF: 1:20-1:100

Properties

Stability & Storage:	Store at 2°C-8°C for 1 month. Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles. Preservative-Free.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	Recombinant Protein: Human BID protein (TMPY-02027)
Antigen Species:	Human
Synonyms:	AI875481;2700049M22Rik;BH3 interacting domain death agonist;AU022477

Research 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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