Data Sheet (Cat.No.TMAY-00868)

TargetMol

Anti-BID Antibody (1O729)

Product Details

Ig Type:	Mouse IgG2a
Reactivity:	Human
Conjugation:	Unconjugated
Clone:	1O729
Purification:	Protein A

Applications

Verified Activity:	Anti-BID mouse monoclonal antibody at 1:500 dilution. -Lane A: Jurkat Whole Cell lysate. -Lysates/proteins at 30 µg per lane. -Secondary -Goat Anti-Mouse IgG H&L (Dylight800) at 1/15000 dilution. -Developed using the Odyssey technique. -Performed under reducing conditions. -Predicted band size:22 kDa. -Observed band size:22 kDa
Application:	ELISA,ELISA(Det),WB
Recommended	WB: 1:500-1:2000; ELISA: 1:1000-1:2000; ELISA(Det): 1:1000-1:10000

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:	BH3 interacting domain death agonist;2700049M22Rik;AI875481;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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