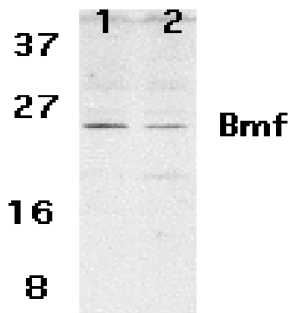
Bmf (NT) Polyclonal Antibody

Product Specifications

Catalog Number:	905-184
Host:	Rabbit
Species Reactivity:	Human and mouse
Applications: *The optimal dilution for a specific application must be determined by the investigator*	**WB:** 2 µg /mL HepG2 or 293 cell lysates can be used for positive control
Predicted m.w:	~25 kDa
Concentration:	See product label
Purification:	Affinity Purified
Format:	PBS, with 0.02% sodium azide
Storage: *Shipping conditions may differ from the recommended storage temperature*	Store at -20˚C
Immunogen:	Synthetic peptide derived from human Bmf1; sequence differs from that of mouse by one amino acid.
Related Products:	
SAB-300	Goat anti-Rabbit IgG Polyclonal Antibody, HRP Conjugate
900-133	Bcl-2 (human total) EIA Kit
AAM-072	Bcl-2 Monoclonal Antibody (83-8B)
AAS-070	Bcl-2 Polyclonal Antibody
AAP-020	Bad Polyclonal Antibody
905-638	Bad Monoclonal Antibody (5E6)



Western blot analysis of human HepG2 (1) and 293 (2) cell lysates, probed with anti-Bmf at 2 µg /mL

Background:

Apoptosis is related to many diseases and development. Members of the Bcl-2 family are critical regulators of apoptosis by either inhibiting or promoting cell death. Bcl-2 homology 3 (BH3) domain is a potent death domain. BH3-only proteins, including Bad, Bid, Bik, Hrk, Bim, Noxa, and PUMA, form a growing subclass of the Bcl-2 family. Bcl-2-modifying factor (Bmf) is a member of the BH3-only protein family that is constitutively expressed in many tissues of both humans and mice[1-2]. The BH3 domain in Bmf is required both for binding to Bcl-2 proteins and for triggering apoptosis. In healthy cells, Bmf associates with the dynein light chain 2 (DLC2) component of the myosin V motors, and is sequestered by the cell's actin cytoskeleton. Disruption of the actin cytoskeleton, either by depolymerization of actin filaments or by detachment of cells from the extracellular matrix, triggers release and activation of Bmf, initiating the downstream apoptotic program[1,2].

References:

1. Puthalakath, H. *et al*. Science (2001) **293**, 1829-1832.
2. Hunt, A. *et al*. Science (2001) **293**, 1784-1785.

5777 Hines Drive • Ann Arbor, MI • 48108 | Tel: 800-833-8651 or 800-668-6113 | Fax: 734-668-2793
www.assaydesigns.com | orders@assaydesigns.com | technical@assaydesigns.com

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