10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

BNIP3L BH3 Domain Antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP1320a

Product Information

Application	IHC-P, E
Primary Accession	O60238
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB3383
Calculated MW	23930
Antigen Region	122-152

Additional Information

Gene ID	665
Other Names	BCL2/adenovirus E1B 19 kDa protein-interacting protein 3-like, Adenovirus E1B19K-binding protein B5, BCL2/adenovirus E1B 19 kDa protein-interacting protein 3A, NIP3-like protein X, NIP3L, BNIP3L, BNIP3A, BNIP3H, NIX
Target/Specificity	This BNIP3L BH3 Domain antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 122~152 amino acids within aa 100-150 (BH3 domain) of human BNIP3L.
Dilution	IHC-P~~1:100~500 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	BNIP3L BH3 Domain Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	BNIP3L
Synonyms	BNIP3A, BNIP3H, NIX
Function	Induces apoptosis. Interacts with viral and cellular anti- apoptosis proteins.

	Can overcome the suppressors BCL-2 and BCL-XL, although high levels of BCL-XL expression will inhibit apoptosis. Inhibits apoptosis induced by BNIP3. Involved in mitochondrial quality control via its interaction with SPATA18/MIEAP: in response to mitochondrial damage, participates in mitochondrial protein catabolic process (also named MALM) leading to the degradation of damaged proteins inside mitochondria. The physical interaction of SPATA18/MIEAP, BNIP3 and BNIP3L/NIX at the mitochondrial outer membrane regulates the opening of a pore in the mitochondrial double membrane in order to mediate the translocation of lysosomal proteins from the cytoplasm to the mitochondrial matrix. May function as a tumor suppressor.
Cellular Location	Nucleus envelope. Endoplasmic reticulum. Mitochondrion outer membrane. Membrane; Single-pass membrane protein. Note=Colocalizes with SPATA18 at the mitochondrion outer membrane

Background

BNIP3L is a member of the BCL2/adenovirus E1B 19 kd-interacting protein (BNIP) family. It interacts with the E1B 19 kDa protein which is responsible for the protection of virally-induced cell death, as well as E1B 19 kDa-like sequences of BCL2, also an apoptotic protector. The protein encoded by this gene is a functional homolog of BNIP3, a proapoptotic protein. This protein may function simultaneously with BNIP3 and may play a role in tumor suppression.

References

Aerbajinai, W., et al., Blood 102(2):712-717 (2003).
Passer, B.J., et al., Proc. Natl. Acad. Sci. U.S.A. 100(5):2284-2289 (2003).
Ohi, N., et al., Cell Death Differ. 6(4):314-325 (1999).
Chen, G., et al., J. Biol. Chem. 274(1):7-10 (1999).
Yasuda, M., et al., Cancer Res. 59(3):533-537 (1999).

Images

Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

Citations

- BNIP3L/Nix-induced mitochondrial fission, mitophagy, and impaired myocyte glucose uptake are abrogated by PRKA/PKA phosphorylation
- Pyruvate kinase [removed]PKM1 and PKM2) in cancer-associated fibroblasts drives stromal nutrient production and tumor growth.
- Autophagy in cancer associated fibroblasts promotes tumor cell survival: Role of hypoxia, HIF1 induction and NFκB activation in the tumor stromal microenvironment.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.