

BNIP3L BH3 Domain Antibody
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP1320a

Specification

BNIP3L BH3 Domain Antibody - Product Information

| | |
|-------------------|------------------------|
| Application | IHC-P,E |
| Primary Accession | O60238 |
| Reactivity | Human, Mouse |
| Host | Rabbit |
| Clonality | Polyclonal |
| Isotype | Rabbit IgG |
| Antigen Region | 122-152 |

BNIP3L BH3 Domain Antibody - 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~~1:50~100

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.

BNIP3L BH3 Domain Antibody - 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

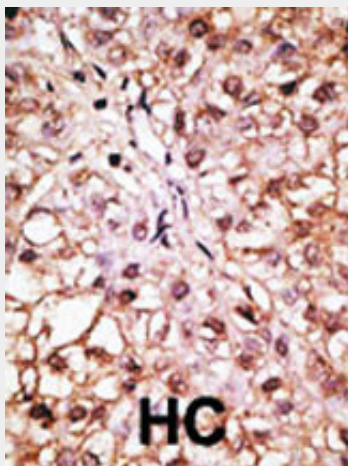
Nucleus envelope. Endoplasmic reticulum. Mitochondrion outer membrane. Membrane; Single-pass membrane protein. Note=Colocalizes with SPATA18 at the mitochondrion outer membrane

BNIP3L BH3 Domain Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

BNIP3L BH3 Domain Antibody - 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.

BNIP3L BH3 Domain Antibody - 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.

BNIP3L BH3 Domain Antibody - 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).

BNIP3L BH3 Domain Antibody - 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 NFkB activation in the tumor stromal microenvironment.](#)