

BNIP3L/Nix Antibody

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications: W, IP	Reactivity: H M	Sensitivity: Endogenous	MW (kDa): 38, 80	Source/Isotype: Rabbit	UniProt ID: #O60238	Entrez-Gene Id: 665
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Product Usage Information**Application**

Western Blotting
Immunoprecipitation

Dilution

1:1000
1:100

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at -20°C. Do not aliquot the antibody.

Specificity/Sensitivity

BNIP3L/Nix Antibody recognizes endogenous levels of total BNIP3L/Nix protein.

Species predicted to react based on 100% sequence homology

Rat

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Glu128 of human BNIP3L/Nix protein. Antibodies are purified by protein A and peptide affinity chromatography.

Background

BCL2/Adenovirus E1B 19 kDa protein-interacting protein 3-like (BNIP3L) (1), also termed BNIP3a (2), B5 (3), and Nix (4), is a member of the Bcl-2 family of apoptotic regulators with highest homology to BNIP3. BNIP3L can bind BNIP3, Bcl-x_L, and Bcl-2 (1-5). BNIP3L forms homodimers that withstand denaturing by SDS and reducing conditions (5). BNIP3L is a mitochondrial protein and knockout studies suggest that BNIP3L regulates autophagic clearance of damaged mitochondria during erythroid maturation via mitochondrial autophagy (6,7). It has been shown that the expression of BNIP3L is up-regulated during terminal erythroid differentiation (6-8), as well as in tumor cell lines during hypoxia (9-11). BNIP3L directly regulates the elimination of mitochondria through its ability to bind to and recruit important components of the autophagic machinery, including LC3/Atg8 and GABARAP proteins, via its amino-terminal LC3-interacting region (LIR) (12). BNIP3L may also indirectly activate phagophore formation either via the recruitment of autophagy proteins or by binding Bcl-x_L, which in turn releases Beclin-1 (13). BNIP3L/Nix also plays a pivotal role in Parkin-mediated mitochondrial autophagy via its ability to mediate the mitochondrial translocation of Parkin (14). Activated BNIP3L can promote the opening of mitochondrial permeability transition pores resulting in mitochondrial depolarization, generation of reactive oxygen species, and induction of necrosis. Due to its involvement in cell death and autophagy, research scientists have implicated BNIP3L in heart disease and cancer (13).

Background References

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5. Imazu, T. et al. (1999) *Oncogene* 18, 4523-9.
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7. Sandoval, H. et al. (2008) *Nature* 454, 232-5.
8. Aerbajinai, W. et al. (2003) *Blood* 102, 712-7.
9. Sowter, H.M. et al. (2001) *Cancer Res* 61, 6669-73.
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11. Fei, P. et al. (2004) *Cancer Cell* 6, 597-609.
12. Novak, I. et al. (2010) *EMBO Rep* 11, 45-51.
13. Zhang, J. and Ney, P.A. (2009) *Cell Death Differ* 16, 939-46.
14. Ding, W.X. et al. (2010) *J Biol Chem* 285, 27879-90.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key**W:** Western Blotting **IP:** Immunoprecipitation**Cross-Reactivity Key****H:** Human **M:** Mouse**Trademarks and Patents**

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