

BNIP3L Antibody / BCL2/adenovirus E1B 19 kDa-interacting protein 3-like (FY13212)

Catalog No.	Formulation	Size
FY13212	Adding 0.2 ml of distilled water will yield a concentration of 500 ug/ml	100 ug

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Availability	1-2 days
Species Reactivity	Human, Mouse, Rat
Format	Lyophilized
Clonality	Polyclonal (rabbit origin)
Isotype	Rabbit IgG
Purity	Immunogen affinity purified
Buffer	Each vial contains 4 mg Trehalose, 0.9 mg NaCl, 0.2 mg Na ₂ HPO ₄ .
UniProt	O60238
Applications	Western Blot : 0.25-0.5ug/ml Immunocytochemistry/Immunofluorescence : 5ug/ml Flow Cytometry : 1-3ug/million cells
Limitations	This BNIP3L antibody is available for research use only.

Description

BNIP3L antibody detects BCL2/adenovirus E1B 19 kDa-interacting protein 3-like, a mitochondrial protein involved in apoptosis, mitophagy, and hypoxia adaptation. The UniProt recommended name is BCL2/adenovirus E1B 19 kDa-interacting protein 3-like (BNIP3L). Also known as NIX, this pro-apoptotic factor serves as a critical mediator of mitochondrial quality control by promoting selective autophagic removal of damaged mitochondria.

Functionally, BNIP3L antibody identifies a 263-amino-acid integral membrane protein localized to the outer mitochondrial membrane. BNIP3L interacts with BCL2 family members and contains an LC3-interacting region (LIR) motif that recruits autophagosomes to mitochondria during mitophagy. Under hypoxic conditions, BNIP3L expression is induced by hypoxia-inducible factor 1-alpha (HIF1A), initiating mitophagy and limiting reactive oxygen species accumulation. This process is essential for maintaining mitochondrial integrity and metabolic adaptation during low oxygen stress.

The BNIP3L gene is located on chromosome 8p21.2 and is expressed in heart, skeletal muscle, and brain. Its transcriptional regulation integrates stress and energy signals from pathways including HIF1A, FOXO3, and PGC1A, linking mitochondrial turnover to cellular homeostasis and survival.

Pathologically, BNIP3L plays dual roles in cell fate: promoting mitophagy and survival under transient stress, but triggering apoptosis under severe hypoxia or damage. Dysregulation contributes to cardiomyopathy, neurodegeneration, and tumor resistance to therapy. In cancer, elevated BNIP3L expression enhances mitochondrial clearance, enabling metabolic flexibility in hypoxic tumor microenvironments. Research using BNIP3L antibody supports studies in apoptosis, autophagy, and mitochondrial signaling.

BNIP3L antibody is validated for western blotting, immunofluorescence, and immunohistochemistry to detect mitophagy regulators. NSJ Bioreagents provides BNIP3L antibody reagents optimized for research in hypoxia, mitochondrial biology, and programmed cell death.

Structurally, BCL2/adenovirus E1B 19 kDa-interacting protein 3-like contains a C-terminal transmembrane domain for mitochondrial localization, a BH3-like domain mediating interactions with BCL2 family proteins, and an LIR motif for autophagosome binding. This antibody enables exploration of BNIP3L's role in balancing mitophagy and apoptosis during cellular stress.

Application Notes

Optimal dilution of the BNIP3L antibody should be determined by the researcher.

Immunogen

A synthetic peptide corresponding to a sequence in the middle region of human BNIP3L was used as the immunogen for the BNIP3L antibody.

Storage

After reconstitution, the BNIP3L antibody can be stored for up to one month at 4°C. For long-term, aliquot and store at -20°C. Avoid repeated freezing and thawing.