## **Recombinant Human BNIP3L Protein**

Catalog Number: PKSH030825



Note: Centrifuge before opening to ensure complete recovery of vial contents.

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SynonymsBNIP3a;NIXSpeciesHumanExpression HostE.coli

SequenceSer 2-Lys 187AccessionQ7Z465-1Calculated Molecular Weight20.4 kDaObserved molecular weight36 kDaTagNone

### **Properties**

**Purity** > 88 % as determined by reducing SDS-PAGE.

**Endotoxin** Please contact us for more information.

**Storage** Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to

-80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots

of reconstituted samples are stable at < -20°C for 3 months.

**Shipping** This product is provided as lyophilized powder which is shipped with ice packs.

**Formulation** Lyophilized from sterile 50mM Tris, 150mM NaCl, 1mM DTT, pH 8.0

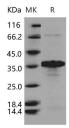
Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as

protectants before lyophilization.

Please refer to the specific buffer information in the printed manual.

**Reconstitution** Please refer to the printed manual for detailed information.

#### Data



> 88 % as determined by reducing SDS-PAGE.

### **Background**

The deletion of BNIP3L results in retention of mitochondria during lens fiber cell remodeling, and that deletion of BNIP3L also results in the retention of endoplasmic reticulum and Golgi apparatus. BNIP3L localizes to the endoplasmic reticulum and Golgi apparatus of wild-type newborn mouse lenses and is contained within mitochondria, endoplasmic reticulum and Golgi apparatus isolated from adult mouse liver. As the cells become packed with keratin bundles, Bnip3L expression triggers mitophagy to rid the cells of the last remaining 'living' characteristic, thus completing the march from 'living' to 'dead' within the hair follicle. during retinal development tissue hypoxia triggers HIF1A/HIF-1 stabilization, resulting in increased expression of the mitophagy receptor BNIP3L/NIX. BNIP3L-dependent mitophagy results in a metabolic shift toward glycolysis essential for RGC neurogenesis. BNIP3L could be a potential therapeutic target for

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