# Recombinant Human BNIP3L Protein

**Catalog Number: PKSH030825**

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

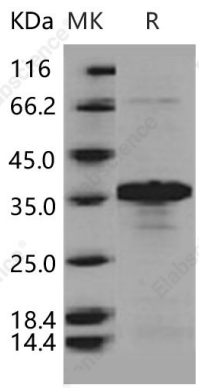
## Description

| | |
|---|---|
| **Species** | Human |
| **Source** | E.coli-derived Human BNIP3L protein Ser 2-Lys 187 |
| **Calculated MW** | 20.4 kDa |
| **Observed MW** | 36 kDa |
| **Accession** | Q7Z465-1 |
| **Bio-activity** | Not validated for activity |

## 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 50 mM Tris, 150 mM NaCl, 1 mM DTT, pH 8.0<br>Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.<br>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 BNIP3 L results in retention of mitochondria during lens fiber cell remodeling, and that deletion of BNIP3 L also results in the retention of endoplasmic reticulum and Golgi apparatus. BNIP3 L 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, Bnip3 L 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 BNIP3 L/NIX. BNIP3 L-dependent mitophagy results in a metabolic shift toward glycolysis essential for RGC neurogenesis. BNIP3 L could be a potential therapeutic target for ischemic stroke


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