Recombinant Human BNIP3L Protein

Catalog Number: PKSH030825

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

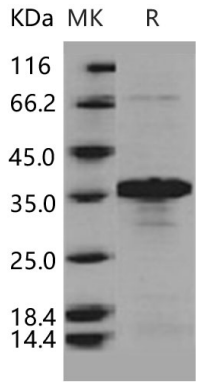
Description

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 50mM Tris, 150mM NaCl, 1mM DTT, pH 8.0 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 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 ischemic stroke

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