

Recombinant Human BLNK/Ly-57 Protein (His Tag)

Catalog Number: PKSH030792

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

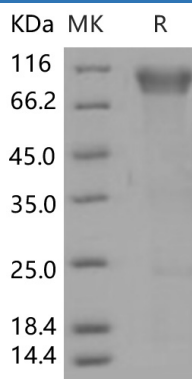
Description

Species	Human
Source	HEK293 Cells-derived Human BLNK/Ly-57 protein Met 1-Ser 456, with an N-terminal His
Calculated MW	53.0 kDa
Observed MW	95-100 kDa
Accession	AAH18906.1
Bio-activity	Measured by its ability to bind human BTK in a functional ELISA.

Properties

Purity	> 90 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per µg of the protein as determined by the LAL method.
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 PBS, pH 7.4 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



> 90 % as determined by reducing SDS-PAGE.

Background

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B-cell linker protein, also known as B-cell adapter containing a SH2 domain protein, B-cell adapter containing a Src homology 2 domain protein, Cytoplasmic adapter protein, Src homology 2 domain-containing leukocyte protein of 65 kDa, SLP-65 and BLNK, is a cytoplasm and cell membrane protein which contains one SH2 domain. BLNK is expressed in B-cell lineage and fibroblast cell lines. Highest levels of expression is in the spleen, with lower levels in the liver, kidney, pancreas, small intestines and colon. BLNK functions as a central linker protein that bridges kinases associated with the B-cell receptor (BCR) with a multitude of signaling pathways, regulating biological outcomes of B-cell function and development. BLNK plays a role in the activation of ERK / EPHB2, MAP kinase p38 and JNK. BLNK modulates AP1 activation. It is important for the activation of NF-kappa-B and NFAT. BLNK plays an important role in BCR-mediated PLCG1 and PLCG2 activation and Ca²⁺ mobilization and is required for trafficking of the BCR to late endosomes. BLNK may be required for the RAC1-JNK pathway. It plays a critical role in orchestrating the pro-B cell to pre-B cell transition. BLNK also plays an important role in BCR-induced B-cell apoptosis. Defects in BLNK are the cause of agammaglobulinemia type 4 (AGM4) which is a primary immunodeficiency characterized by profoundly low or absent serum antibodies and low or absent circulating B cells due to an early block of B-cell development.

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