Recombinant Human BLK Protein (GST Tag)

Catalog No. PKSH030381

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

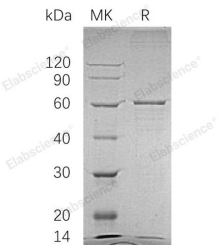
Description

Synonyms	Tyrosine-Protein Kinase Blk;B Lymphocyte Kinase;p55-Blk;BLK;MODY11
Species	Human
Expression Host	Baculovirus-Insect Cells
Sequence	Met 1-Pro 505
Accession	NP_001706.2
Calculated Molecular Weight	84 kDa
Observed molecular weight	84 kDa
Tag	N-GST
Bioactivity	The specific activity was determined to be 17.4 nmol/min/mg using Poly(Glu, Tyr)4:1 peptide as substrate.

Properties

Purity	> 88 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < - 20°C.
Formulation	Supplied as sterile solution of 20mM Tris, 500mM NaCl, 5mM GSH, pH 7.4
Reconstitution	Not Applicable

Data



> 88 % as determined by reducing SDS-PAGE.

Background

Tyrosine-protein kinase Blk, also known as B lymphocyte kinase, p55-Blk and BLK, is a member of theprotein kinase superfamily, Tyr protein kinase family and SRC subfamily. BLK / p55-Blk is expressed in lymphatic organs, pancreatic islets, Leydig cells, striate ducts of salivary glands and hair follicles. BLK / p55-Blk is a src-family protein tyrosine kinase specifically expressed in B-lineage cells of mice. The early onset of Blk expression during B-cell development in the bone marrow and the high expression levels of Blk in mature B cells suggest a possible important role of Blk in B-cell

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physiology. It is a modulator of beta-cells function, acting through the up-regulation of PDX1 and NKX6-1 and consequent stimulation of insulin secretion in response to glucose. Defects in BLK are a cause of maturity-onset diabetes of the young type 11 which is a form of diabetes that is characterized by an autosomal dominant mode of inheritance, onset in childhood or early adulthood (usually before 25 years of age), a primary defect in insulin secretion and frequent insulin-independence at the beginning of the disease.

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