

BLK Protein, Human, Recombinant (GST)

General Information

Synonyms:	MODY11;BLK proto-oncogene, Src family tyrosine kinase
Protein Construction:	A DNA sequence encoding the human BLK (NP_001706.2) (Met 1-Pro 505) was fused with the GST tag at the N-terminus. Predicted N terminal: Met
Species:	Human
Expression Host:	Baculovirus Insect Cells
Accession:	P51451
Molecular Weight:	84 kDa (predicted); 84 kDa (reducing conditions)

QC Testing

Biological Activity:	The specific activity was determined to be 17.4 nmol/min/mg using Poly(Glu,Tyr)4:1 peptide as substrate.
Purity:	> 88 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU/μg of the protein as determined by the LAL method.
Formulation:	Supplied as sterile 20 mM Tris, 500 mM NaCl, 5 mM GSH, pH 7.4.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store the product under sterile conditions at -20°C to -80°C. Samples are stable for up to 12 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

Proteins are shipped with blue ice.

Protein Background

Tyrosine-protein kinase Blk, also known as B lymphocyte kinase, p55-Blk and BLK, is a member of the protein 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 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

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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.

Reference

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Texido, G. et al., 2000, Mol Cell Biol. 20 (4):1227-33.
Borowiec M. et al., 2009, Proc. Natl. Acad. Sci. USA. 106: 14460-5.

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