Recombinant Protein Technical Manual

Recombinant Human BLK Protein (GST Tag)(Active)

RPES1476

Product Data:

Product SKU: RPES1476

Size: 20µg

Species: Human

Expression host: Baculovirus-Insect Cells

Uniprot: NP_001706.2

Protein Information:

Molecular Mass:

AP Molecular Mass: 84 kDa

Tag: N-GST

Bio-activity: The specific activity was determined to be 17.4 nmol/min/mg using Poly(Glu,Tyr)4:1 peptide as substrate.

Purity: 90 % 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 20mM Tris, 300mM NaCl, 0.5mM GSH, pH 7.5, 25% glycerol.

Reconstitution: Please refer to the printed manual for detailed information.

Application:

Synonyms: Tyrosine-Protein Kinase Blk; B Lymphocyte Kinase; p55-Blk; BLK;MODY11

Immunogen Information:

Sequence: Met 1-Pro 505

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 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.