

Mer Protein, Human, Recombinant (His & GST)

General Information

Synonyms:	MER proto-oncogene, tyrosine kinase;c-Eyk;RP38;c-mer;Tyro12;MER
Protein Construction:	A DNA sequence encoding the human MERTK (Q12866) protein kinase domain (Glu 578-Tyr 872) was fused with the N-terminal polyhistidine-tagged GST tag at the N-terminus. Predicted N terminal: Met
Species:	Human
Expression Host:	Baculovirus Insect Cells
Accession:	Q12866
Molecular Weight:	62 kDa (predicted); 50 kDa (reducing conditions)

QC Testing

Biological Activity:	Kinase activity untested
Purity:	> 92 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU/μg of the protein as determined by the LAL method.
Formulation:	Supplied as sterile 50 mM Tris, 100 mM NaCl, pH 7.4, 20% gly, 0.3 mM DTT.

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

Proto-oncogene tyrosine-protein kinase MER (MERTK) is a member of the MER/AXL/TYRO3 receptor kinase family and encodes a transmembrane protein with two fibronectin type-III domains, two Ig-like C2-type (immunoglobulin-like) domains, and one tyrosine kinase domain. MERTK is localized in the membrane and is not expressed in normal B- and T-lymphocytes but is expressed in numerous neoplastic B- and T-cell lines. This protein is highly expressed in the testis, ovary, prostate, lung, and kidney, with lower expression in the spleen, small intestine, colon, and liver. MERTK regulates many physiological processes including cell survival, migration, differentiation, and phagocytosis of apoptotic cells (efferocytosis). Ligand binding at the cell surface induces autophosphorylation of MERTK on its intracellular domain that provides docking sites for downstream signaling

molecules. MERTK signaling plays a role in various processes such as macrophage clearance of apoptotic cells, platelet aggregation, cytoskeleton reorganization, and engulfment. MERTK plays also an important role in the inhibition of Toll-like receptors (TLRs)-mediated innate immune response by activating STAT1, which selectively induces the production of suppressors of cytokine signaling SOCS1 and SOCS3. Defects in MERTK are the cause of retinitis pigmentosa type 38.

Reference

Thompson DA, et al. (2002) Retinal dystrophy due to paternal isodisomy for chromosome 1 or chromosome 2, with homoallelism for mutations in RPE65 or MERTK, respectively. *Am J Hum Genet.* 70 (1): 224-9.

Tada A, et al. (2006) Screening of the MERTK gene for mutations in Japanese patients with autosomal recessive retinitis pigmentosa. *Mol Vis.* 12: 441-4.

McHenry CL, et al. (2004) MERTK arginine-844-cysteine in a patient with severe rod-cone dystrophy: loss of mutant protein function in transfected cells. *Invest Ophthalmol. Vis Sci.* 45 (5): 1456-63.

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