Recombinant Protein Technical Manual

Recombinant Mouse DDR1 Kinase/MCK10 Protein (His &GST Tag)(Active)

RPES1894

Product Data:

Product SKU: RPES1894

Size: 20µg

Species: Mouse

Expression host: Baculovirus-Insect Cells

Uniprot: Q03146-2

Protein Information:

Molecular Mass:	75.8 kDa
AP Molecular Mass:	68 kDa
Tag:	N-His-GST
Bio-activity:	The specific activity was determined to be 2 nmol/min/mg using synthetic modified AXLtide peptide (modified-CKKSRGDYMTMQIG) as substrate.
Purity:	> 95 % as determined by 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, 500mM NaCl, pH 7.4, 10% glycerol, 2mM DTT
Reconstitution:	Please refer to the printed manual for detailed information.
Application:	
Synonyms:	6030432F18;AI323681;Cak;CD167a;Nep;PTK3A

Immunogen Information:

Sequence: Leu444-Val874

Background:

Discoidin domain receptor family, member 1 (DDR1), also known as or CD167a (cluster of differentiation 167a), and Mammary carcinoma kinase 10 (MCK10), belongs to a subfamily of tyrosine kinase receptors with an extracellular domain homologous to Dictyostellium discoideum protein discoidin 1. Receptor tyrosine kinases play a key role in the communication of cells with their microenvironment. These kinases are involved in the regulation of cell growth, differentiation and metabolism. Expression of DDR1/MCK10/CD167 is restricted to epithelial cells, particularly in the kidney, lung, gastrointestinal tract, and brain. In addition, it has been shown to be significantly overexpressed in several human tumors. DDR1/MCK10/CD167 plays an important role in regulating attachment to collagen, chemotaxis, proliferation, and MMP production in smooth muscle cells. DDR1 functions in a feedforward loop to increase p53 levels and at least some of its effectors. Inhibition of DDR1 function resulted in strikingly increased apoptosis of wild-type p53-containing cells in response to genotoxic stress through a caspase-dependent pathway.