Recombinant Human DDR1 Kinase/MCK10 Protein (aa 444-913, His &GST Tag)

Catalog Number: PKSH030389

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

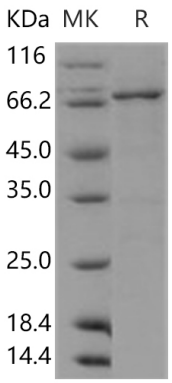
Description

Species	Human
Source	Baculovirus-Insect Cells-derived Human DDR1 Kinase/MCK10 protein Arg444-Val913, with an N-terminal His & GST
Calculated MW	80.0 kDa
Observed MW	80 kDa
Accession	Q08345-1
Bio-activity	The specific activity was determined to be 2. 75 nmol/min/mg using synthetic AXLtide peptide(CKKSRGDYMTMQIG) as substrate.

Properties

Purity	> 89 % as determined by reducing SDS-PAGE.
Concentration	Subject to label value.
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, pH 7.4, 10% glycerol, 3mM DTT

Data



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Background

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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.

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