Recombinant Human MAPK9 Protein (His Tag)

Catalog Number: PDEH101041

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

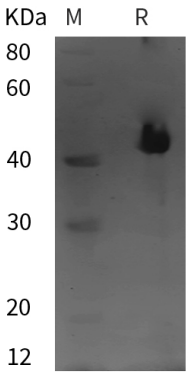
Description

Species	Human
Source	E.coli-derived Human MAPK9 protein Phe101-Arg424, with an N-terminal His & C-terminal His
Calculated MW	35.5 kDa
Observed MW	41 kDa
Accession	P45984
Bio-activity	Not validated for activity

Properties

Purity	> 95% as determined by reducing SDS-PAGE.
Endotoxin	< 10 EU/mg of the protein as determined by the LAL method
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

Data



SDS-PAGE analysis of Human MAPK9 proteins, 2 μg/lane of Recombinant Human MAPK9 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 41 kDa.

Background

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Mitogen-activated protein kinase 9 (MAPK9), also well known as c-Jun N-terminal kinase (JNK2), is a member of MAP kinase subfamily belonging to the protein kinase superfamily. The crystal structure of human JNK2 complexed with an indazole inhibitor by applying a high-throughput protein engineering and surface-site mutagenesis approach. A novel conformation of the activation loop is observed, which is not compatible with its phosphorylation by upstream kinases. This activation inhibitory conformation of JNK2 is stabilized by the MAP kinase insert that interacts with the activation loop in an induced-fit manner. It suggest that the MAP kinase insert of JNK2 plays a role in the regulation of JNK2 activation, possibly by interacting with intracellular binding partners. JNK2 deficiency leads to reduced c-Jun degradation, thereby augmenting c-Jun levels and cellular proliferation, and suggests that JNK2 is a negative regulator of cellular proliferation in multiple cell types. JNK2 blocks the ubiquitination of tumor suppressor p53, and thus increases the stability of p53 in nonstressed cells. JNK2 negatively regulates antigen-specific CD8+ T cell expansion and effector function, and thus selectively blocking JNK2 in CD8+ T cells may potentially enhance anti-tumor immune response. Lack of JNK2 expression was associated with higher tumor aneuploidy and reduced DNA damage response. Additionally,the JNK2 protein could be a novel therapeutic target in dry eye disease, and may provide a novel target for prevention of vascular disease and atherosclerosis.

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