

Recombinant Human JNK2/MAPK9 Protein (His Tag)

Catalog Number: PKSH031449

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

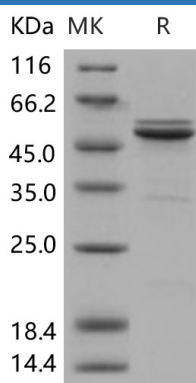
Description

Species	Human
Source	Baculovirus-Insect Cells-derived Human JNK2/MAPK9 protein Met 1-Arg 424, with an C-terminal His
Calculated MW	49.5 kDa
Observed MW	49.5 kDa
Accession	NP_002743.3
Bio-activity	Not validated for activity

Properties

Purity	> 90 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per µg 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 sterile 50mM Tris, 100mM NaCl, pH 8.0, 10% glycerol, 0.5mM EDTA, 0.5mM PMSF Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 90 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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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.