JNK2 Protein, Human (sf9, His)

Cat. No.:	HY-P74794
Synonyms:	Mitogen-activated protein kinase 9; MAPK 9; JNK-55; SAPK1a
Species:	Human
Source:	Sf9 insect cells
Accession:	P45984 (M1-R424)
Gene ID:	5601
Molecular Weight:	Approximately 49.5 kDa

PROPERTIES

Biological Activity	The enzyme activity of this recombinant protein is testing in progress, we cannot offer a guarantee yet.
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of 50 mM Tris, 100 mM NaCl, pH 8.0, 10% Glycerol, 0.5 mM EDTA, 0.5 mM PMSF. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	JNK2, a serine/threonine-protein kinase, is a central player in diverse cellular processes, encompassing cell proliferation, differentiation, migration, transformation, and programmed cell death. Activation of the stress-activated protein kinase/c-Jun N-terminal kinase (SAP/JNK) signaling pathway by extracellular stimuli, such as pro-inflammatory cytokines or physical stress, involves the phosphorylation and activation of JNK2 by dual specificity kinases MAP2K4/MKK4 and MAP2K7/MKK7. Within this cascade, JNK2 phosphorylates critical transcription factors, particularly components of AP-1 like JUN and ATF2, thereby modulating AP-1 transcriptional activity. In response to oxidative or ribotoxic stresses, JNK2 inhibits rRNA synthesis by phosphorylating and inactivating the RNA polymerase 1-specific transcription initiation factor RRN3. Additionally, JNK2 plays a crucial role in stressed cell apoptosis by phosphorylating key regulatory factors, including TP53 and YAP1. In T-cells, JNK2, along with MAPK8, is essential for the polarized differentiation of T-helper cells into Th1 cells. Furthermore, upon T-cell receptor (TCR) stimulation, JNK2 is activated to regulate JUN protein levels by interacting with CARMA1, BCL10, MAP2K7, and MAP3K7/TAK1. JNK2 is also instrumental in osmotic stress-induced disruption of epithelial tight junctions and, when activated, promotes beta-catenin/CTNNB1 degradation, thereby inhibiting the canonical Wnt signaling pathway. Additionally, JNK2 participates in neurite growth in spiral ganglion neurons and phosphorylates the CLOCK-BMAL1

heterodimer, contributing to the regulation of the circadian clock. Notably, JNK2 isoforms exhibit distinct binding patterns, with alpha-1 and alpha-2 preferentially binding to JUN, while beta-1 and beta-2 bind to ATF2. However, phosphorylation efficiency remains consistent across all isoforms, and JUNB is not a substrate for JNK2 alpha-2, with JUND binding only weakly to it.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA