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JNK2 (MAPK9) Antibody (C-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7506A

Product Information

Application	WB, IHC-P, E
Primary Accession	P45984
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	48139
Antigen Region	349-379

Additional Information

Gene ID	5601
Other Names	Mitogen-activated protein kinase 9, MAP kinase 9, MAPK 9, JNK-55, Stress-activated protein kinase 1a, SAPK1a, Stress-activated protein kinase JNK2, c-Jun N-terminal kinase 2, MAPK9, JNK2, PRKM9, SAPK1A
Target/Specificity	This JNK2 (MAPK9) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 349-379 amino acids from the C-terminal region of human JNK2 (MAPK9).
Dilution	WB~~1:1000 IHC-P~~1:100~500 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	JNK2 (MAPK9) Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	MAPK9
Synonyms	JNK2, PRKM9, SAPK1A
Function	Serine/threonine-protein kinase involved in various processes such as cell proliferation, differentiation, migration, transformation and programmed cell

	death (PubMed:10376527, PubMed:15805466, PubMed:17525747, PubMed:19675674, PubMed:20595622, PubMed:21364637, PubMed:22441692, PubMed:34048572). Extracellular stimuli such as pro-inflammatory cytokines or physical stress stimulate the stress- activated protein kinase/c-Jun N-terminal kinase (SAP/JNK) signaling pathway. In this cascade, two dual specificity kinases MAP2K4/MKK4 and MAP2K7/MKK7 phosphorylate and activate MAPK9/JNK2 (PubMed:10376527, PubMed:15805466, PubMed:17525747, PubMed:19675674, PubMed:20595622, PubMed:21364637, PubMed:22441692, PubMed:34048572). In turn, MAPK9/JNK2 phosphorylates a number of transcription factors, primarily components of AP-1 such as JUN and ATF2 and thus regulates AP-1 transcriptional activity (PubMed:10376527). In response to oxidative or ribotoxic stresses, inhibits rRNA synthesis by phosphorylating and inactivating the RNA polymerase 1-specific transcription initiation factor RRN3 (PubMed:15805466). Promotes stressed cell apoptosis by phosphorylating key regulatory factors including TP53 and YAP1 (PubMed:17525747, PubMed:21364637). In T-cells, MAPK8 and MAPK9 are required for polarized differentiation of T-helper cells into Th1 cells (PubMed:19290929). Upon T-cell receptor (TCR) stimulation, is activated by CARMA1, BCL10, MAP2K7 and MAP3K7/TAK1 to regulate JUN protein levels (PubMed:19290929). Plays an important role in the osmotic stress- induced epithelial tight-junctions disruption (PubMed:20595622). When activated, promotes beta-catenin/CTNNB1 degradation and inhibits the canonical Wnt signaling pathway (PubMed:19675674). Also participates in neurite growth in spiral ganglion neurons (By similarity). Phosphorylates the CLOCK-BMAL1 heterodimer and plays a role in the regulation of the circadian clock (PubMed:22441692). Phosphorylates POU5F1, which results in the inhibition of POU5F1's transcriptional activity and enhances its proteasomal degradation (By similarity). Phosphorylates ALKBH5 in response to reactive oxygen species (ROS), promoting ALKBH5 sumoylation and inactivation (PubMed:34048572).
Cellular Location	Cytoplasm. Nucleus. Note=Colocalizes with POU5F1 in the nucleus. {ECO:0000250|UniProtKB:Q9WTU6}

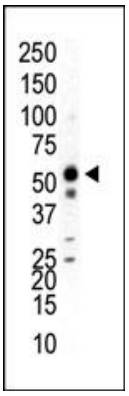
Background

JNK2 responds to activation by environmental stress and pro-inflammatory cytokines by phosphorylating a number of transcription factors, primarily components of AP-1 such as c-Jun and ATF2 and thus regulates AP-1 transcriptional activity. In T-cells, JNK1 and JNK2 are required for polarized differentiation of T-helper cells into Th1 cells. JNK2 isoforms display different binding patterns: alpha-1 and alpha-2 preferentially bind to c-Jun, whereas beta-1 and beta-2 bind to ATF2. However, there is no correlation between binding and phosphorylation, which is achieved at about the same efficiency by all isoforms. JUNB is not a substrate for JNK2 alpha-2, and JUND binds only weakly to it. JNK2 is activated by threonine and tyrosine phosphorylation by either of two dual specificity kinases, MAP2K4 and MAP2K7. It is inhibited by dual specificity phosphatases, such as DUSP1. The protein has been shown to bind to at least three scaffolding proteins, MAPK8IP1/JIP-1, MAPK8IP2/JIP-2 and MAPK8IP3/JIP-3/JSAP1. These proteins also bind other components of the JNK signaling pathway

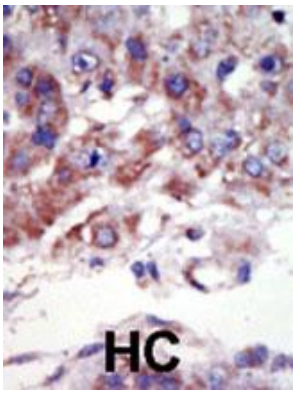
References

Gupta, S., et al., EMBO J. 15(11):2760-2770 (1996).
Sluss, H.K., et al., Mol. Cell. Biol. 14(12):8376-8384 (1994).
Kallunki, T., et al., Genes Dev. 8(24):2996-3007 (1994).
Fleming, Y., et al., Biochem. J. 352 Pt 1, 145-154 (2000).

Images



Western blot analysis of anti-JNK2 Pab (Cat. #AP7506a) in Jurkat cell lysate. JNK2 (arrow) was detected using purified Pab. Secondary HRP-anti-rabbit was used for signal visualization with chemiluminescence.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

Citations

- Isokotomolide A from Cinnamomum kotoense Induce Melanoma Autophagy and Apoptosis
- Regulation of mechanical stress-induced MMP-13 and ADAMTS-5 expression by RUNX-2 transcriptional factor in SW1353 chondrocyte-like cells.