1. Phylogeny:  
   MAPK8, commonly known as JNK1, is a serine/threonine protein kinase that belongs to the Mitogen‐Activated Protein Kinase (MAPK) superfamily, more specifically to the stress‐activated protein kinase (SAPK) subgroup (barr2001thecjunnterminal pages 1-3, kyriakis2012mammalianmapksignal pages 1-2). Within the SAPK branch, JNK1 is closely related to JNK2 and JNK3, with gene duplication and alternative splicing events generating multiple isoforms that are conserved across vertebrate species (herdegen2005contextspecificinhibitionof pages 1-2, manning2003targetingjnkfor pages 2-3). Phylogenetic reconstruction based on comparative kinase genomic analyses shows that the JNK subfamily has maintained its characteristic dual phosphorylation motif through evolution, likely descending from an ancestral MAPK present in the Last Eukaryotic Common Ancestor (liu2016aconservedmotif pages 1-2, li2011evolutionaryhistoryof pages 11-12). This evolutionary conservation is further supported by deep phylogenetic studies that group JNK1 with other MAPKs sharing similar activation loop motifs and catalytic domain organization (orand2023revealingthemechanism pages 175-178, kyriakis2012mammalianmapksignal pages 2-3).
2. Reaction Catalyzed:  
   MAPK8/JNK1 catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of serine or threonine residues in target proteins. The overall reaction is as follows: ATP + [protein]–(L-serine or L-threonine) → ADP + [protein]–(phospho-L-serine/threonine) + H⁺ (barr2001thecjunnterminal pages 1-3).
3. Cofactor Requirements:  
   The kinase activity of MAPK8/JNK1 requires divalent metal ions, most notably Mg²⁺, which facilitates the binding and proper orientation of ATP in the catalytic cleft (barr2001thecjunnterminal pages 1-3).
4. Substrate Specificity:  
   MAPK8/JNK1 is a serine/threonine kinase that preferentially phosphorylates substrates containing serine or threonine residues immediately followed by a proline (Ser/Thr-Pro motifs). This preference is exemplified by its phosphorylation of the transcription factor c-Jun at Ser63 and Ser73, leading to modulation of AP-1 transcriptional activity (barr2001thecjunnterminal pages 1-3, bogoyevitch2006usesforjnk pages 1-2). Recent comprehensive analyses of kinase substrate specificities have polished the consensus motif for human serine/threonine kinases to an extended model that includes flanking basic residues in some contexts (Johnson2023Atlas pages 1-?); while for tyrosine kinases the intrinsic substrate specificity has been mapped as well, although these data are less directly applicable to MAPK8/JNK1 (Yaron-Barir2024 pages 1-?). Therefore, for JNK1 the core determinant is the proline-directed motif, with additional context provided by docking interactions that enhance specificity toward substrates such as JUN, ATF2, and others (davis2000signaltransductionby pages 2-3, herdegen2005contextspecificinhibitionof pages 2-4).
5. Structure:  
   MAPK8/JNK1 adopts the typical bilobal structure characteristic of MAP kinases. Its central kinase domain is divided into an N-terminal lobe composed primarily of β‑sheets and a C-terminal lobe dominated by α‑helices. The active site is located in the cleft between these lobes and contains a highly conserved ATP-binding pocket, a catalytic loop, and an activation loop that harbors the Thr-Pro-Tyr (T-P-Y) motif, which must undergo dual phosphorylation for full catalytic activation (heo2004structuralbasisfor pages 1-2, orand2023revealingthemechanism pages 33-38). This kinase also contains a conserved docking groove that facilitates the interaction with specific short linear motifs (SLiMs) in substrate proteins and scaffold proteins such as JIP1; these interactions modulate substrate binding and improve catalytic efficiency (bogoyevitch2006usesforjnk pages 2-3, heo2004structuralbasisfor pages 11-11). Additional structural features include the hydrophobic spine and the C-helix, which are essential for maintaining the active conformation of the kinase and its proper allosteric regulation (orand2023revealingthemechanism pages 41-45).
6. Regulation:  
   MAPK8/JNK1 is regulated primarily through phosphorylation by dual-specificity MAPK kinases, specifically MKK4 and MKK7, which phosphorylate the Tyr185 and Thr183 residues in its activation loop, respectively (ansideri2018multiplestrategiestargeting pages 38-42, park2019mkk7theessential pages 1-2). In addition to this dual phosphorylation, JNK1 activity is modulated by scaffold proteins, such as members of the JIP family, that spatially and temporally coordinate the assembly of the upstream kinases (MKKKs and MAP2Ks) with JNK1 and its substrates (johnson2007thecjunkinasestressactivated pages 5-6, orand2023revealingthemechanism pages 175-178). Negative regulation is mediated by dual-specificity phosphatases (DUSPs) that dephosphorylate JNK1 and, consequently, downregulate its signaling output (ha2019phosphorylationdynamicsof pages 6-7, zeke2016jnksignalingregulation pages 1-2). Under conditions of cellular stress, such as oxidative stress or UV irradiation, additional regulatory inputs including ubiquitination and caspase-mediated cleavage may occur, which can either enhance or attenuate JNK1 signaling (silva2005mixedlineagekinase–c‐jun pages 2-3, yan2024theroleof pages 7-8).
7. Function:  
   MAPK8/JNK1 plays diverse roles in cellular processes and signal transduction pathways. It is a key mediator in the SAPK/JNK signaling cascade, where its activation leads to the phosphorylation of numerous downstream substrates including transcription factors such as c-Jun, ATF2, and JDP2; these phosphorylation events modulate gene expression in response to extracellular stress stimuli such as pro-inflammatory cytokines and physical stress (ansideri2018multiplestrategiestargeting pages 38-42, barr2001thecjunnterminal pages 1-3). In addition, MAPK8/JNK1 phosphorylates substrates involved in cell cycle regulation and DNA replication, such as the replication licensing factor CDT1; this phosphorylation disrupts the interaction between CDT1 and histone acetylase HBO1, thereby inhibiting replication initiation (ansideri2018multiplestrategiestargeting pages 38-42). JNK1 also promotes apoptosis under prolonged stress by phosphorylating regulators including p53 and YAP1 (ansideri2018multiplestrategiestargeting pages 38-42). In T-cells, both JNK1 and the related kinase MAPK9 are required for the proper polarization of T-helper cells into Th1 cells, indicating a role in adaptive immunity (bogoyevitch2004targetingthejnk pages 1-2). Moreover, MAPK8/JNK1 contributes to cell survival signals in erythroid cells by phosphorylating the pro-apoptotic protein BAD following erythropoietin (EPO) stimulation (ansideri2018multiplestrategiestargeting pages 38-42). Additional functions include modulation of autophagy through phosphorylation of BCL2, regulation of microtubule dynamics via phosphorylation of STMN2, and even involvement in the regulation of the circadian clock by targeting the CLOCK-BMAL1 heterodimer (ansideri2018multiplestrategiestargeting pages 38-42, yan2024theroleof pages 2-4). These activities underscore the central role of JNK1 in transducing stress signals and modulating a diverse array of cellular responses (davis2000signaltransductionby pages 1-2, mehan2011jnkastressactivated pages 2-4).
8. Other Comments:  
   Several inhibitors of MAPK8/JNK1 have been identified, including ATP-competitive molecules such as SP600125 and novel reversible covalent inhibitors that target specific cysteine residues outside the conserved active site (balint2024reversiblecovalentcjun pages 17-18, sammons2018unconventionalapproachesto pages 17-20). Although these inhibitors have shown promise experimentally, their selectivity compared to other MAPK isoforms remains a challenge due to the high conservation of the ATP-binding pocket (heo2004structuralbasisfor pages 1-2, yan2024theroleof pages 22-23). Dysregulation of MAPK8/JNK1 signaling is implicated in multiple human diseases, including neurodegenerative disorders, cancer, chronic inflammation, autoimmune diseases, and metabolic syndromes (yan2024theroleof pages 1-2, silva2005mixedlineagekinase–c‐jun pages 2-3). Notable disease-associated mutations in JNK1 itself are rarely reported; however, altered upstream regulation via MAP2Ks and failures in dephosphorylation by DUSPs contribute to aberrant signaling linked to these pathologies (ha2019phosphorylationdynamicsof pages 6-7, zeke2016jnksignalingregulation pages 1-2). The ongoing development of more selective inhibitors and allosteric modulators holds therapeutic potential for targeting the specific aspects of JNK1-mediated signaling (balint2024reversiblecovalentcjun pages 17-18).
9. References:  
   ansideri2018multiplestrategiestargeting pages 38-42; balint2024reversiblecovalentcjun pages 17-18; barr2001thecjunnterminal pages 1-3; bogoyevitch2004targetingthejnk pages 1-2; bogoyevitch2004targetingthejnk pages 10-11; bogoyevitch2006usesforjnk pages 1-2; bogoyevitch2006usesforjnk pages 2-3; bogoyevitch2010cjunnterminalkinase pages 10-10; bubici2014jnksignallingin pages 1-2; burke2007inhibitionofmitogenactivated pages 3-5; davis2000signaltransductionby pages 1-2; davis2000signaltransductionby pages 2-3; ha2019phosphorylationdynamicsof pages 3-6; ha2019phosphorylationdynamicsof pages 6-7; heo2004structuralbasisfor pages 1-2; heo2004structuralbasisfor pages 11-11; herdegen2005contextspecificinhibitionof pages 1-2; herdegen2005contextspecificinhibitionof pages 2-4; johnson2007thecjunkinasestressactivated pages 1-2; johnson2007thecjunkinasestressactivated pages 5-6; karin2005fromjnkto pages 3-5; kyriakis2012mammalianmapksignal pages 1-2; kyriakis2012mammalianmapksignal pages 2-3; liu2016aconservedmotif pages 1-2; liu2016aconservedmotif pages 10-10; manning2003targetingjnkfor pages 2-3; mehan2011jnkastressactivated pages 2-4; orand2023revealingthemechanism pages 175-178; orand2023revealingthemechanism pages 25-29; orand2023revealingthemechanism pages 33-38; orand2023revealingthemechanism pages 41-45; orand2023revealingthemechanisma pages 175-178; orand2023revealingthemechanisma pages 22-25; orand2023revealingthemechanisma pages 25-29; orand2023revealingthemechanisma pages 41-45; park2019mkk7theessential pages 1-2; raman2007differentialregulationand pages 4-5; sammons2018unconventionalapproachesto pages 17-20; silva2005mixedlineagekinase–c‐jun pages 2-3; yan2024theroleof pages 1-2; yan2024theroleof pages 2-4; yan2024theroleof pages 22-23; yan2024theroleof pages 23-25; yan2024theroleof pages 4-7; yan2024theroleof pages 7-8; zeke2016jnksignalingregulation pages 1-2; huang2024reconstructingthedeep pages 5-7; li2011evolutionaryhistoryof pages 11-12; Johnson2023Atlas; Yaron-Barir2024.

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