1. Phylogeny  
   Mitogen‐activated protein kinase 8 (MAPK8), also known as the c‐Jun N‐terminal kinase 1 (JNK1), belongs to the evolutionarily conserved mitogen‐activated protein kinase (MAPK) superfamily, which comprises a core set of kinases present from yeast through to humans. JNK1 is one of three JNK isoforms (JNK1/MAPK8, JNK2/MAPK9, and JNK3/MAPK10) that appear to have diverged through gene duplication events in early metazoans; in vertebrates, JNK1 and JNK2 are ubiquitously expressed, whereas JNK3 is largely confined to neuronal tissues, the heart, and testes (johnson2007thecjunkinasestressactivated pages 1-2). In the context of the human kinome, JNK1 is assigned to the stress‐activated protein kinases subgroup within the MAPK family, a group that is characterized by their regulation via dual‐phosphorylation within a Thr–Pro–Tyr (TPY) motif in the activation loop. Phylogenetic analyses indicate that MAPK8 is an ancient kinase that shares a high degree of structural and catalytic conservation with other MAPKs and is part of a highly intertwined signaling network that also includes upstream MAPK kinase kinases (MAP3Ks) and MAPK kinases (MAP2Ks) such as MKK4 and MKK7 (orand2023revealingthemechanism pages 25-29, johnson2007thecjunkinasestressactivated pages 1-2).
2. Reaction Catalyzed  
   MAPK8/JNK1 catalyzes the transfer of a phosphate group from ATP to serine/threonine residues on substrate proteins, thereby producing ADP and the phosphorylated protein along with the release of a proton. In biochemical terms, the reaction can be summarized as follows: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (ansideri2018multiplestrategiestargeting pages 35-38).
3. Cofactor Requirements  
   The catalytic activity of MAPK8/JNK1, like that of most kinases, is dependent on the presence of divalent metal ion cofactors, with Mg²⁺ being essential for the coordination of ATP within the kinase active site. This requirement ensures proper binding and orientation of ATP for efficient phosphoryl transfer to the substrate (cargnello2011activationandfunction pages 5-6).
4. Substrate Specificity  
   MAPK8/JNK1 is a proline-directed serine/threonine kinase that exhibits substrate specificity largely determined by the amino acid context surrounding the phosphorylation site. JNK1 preferentially phosphorylates substrates on serine or threonine residues that are immediately followed by a proline residue (S/T-P motif), a feature that facilitates its targeting of transcription factors such as c-Jun, whose activity is enhanced upon phosphorylation at Ser63 and Ser73. In addition to c-Jun, MAPK8 phosphorylates a wide range of substrates including ATF2, p53, CDT1, and various regulatory proteins involved in apoptosis, microtubule dynamics, circadian rhythm regulation, and inflammatory signaling (johnson2007thecjunkinasestressactivated pages 1-2, bogoyevitch2006usesforjnk pages 2-3, cargnello2011activationandfunction pages 5-6). The consensus motif recognized by JNK1 reflects a requirement for a minimal serine/threonine-proline sequence, although additional docking interactions with substrate docking regions or motifs, such as those provided by JNK-interacting proteins (JIPs), further refine its substrate specificity (zeke2015systematicdiscoveryof pages 7-11).
5. Structure  
   MAPK8/JNK1 is organized into a conserved kinase fold that is characteristic of serine/threonine kinases and displays a bilobal architecture. It contains a small N-terminal lobe predominantly composed of anti-parallel β-sheets that is responsible for ATP binding, and a larger C-terminal lobe enriched in α-helices that is critical for substrate recognition and proper alignment of catalytic residues (rehfeldt2020cjunnterminalkinase pages 11-13, orand2023revealingthemechanism pages 25-29). A key structural feature is the activation loop, which harbors a conserved Thr-Pro-Tyr (TPY) motif; dual phosphorylation at the threonine and tyrosine residues in this motif by the upstream kinases MKK4 and MKK7 is essential for JNK1 activation (ansideri2018multiplestrategiestargeting pages 38-42, barr2001thecjunnterminal pages 3-5). The structure also reveals a glycine-rich loop (G-loop) within the N-terminal domain that interacts with ATP, while the C-helix, often containing a critical lysine residue that forms a salt bridge with a glutamate residue, plays a role in stabilizing the active conformation of the enzyme (rehfeldt2020cjunnterminalkinase pages 11-13, bogoyevitch2006usesforjnk pages 4-6). Unique aspects of JNK1’s structure include well-characterized docking sites—such as the D-recruitment site (DRS)—which mediate interactions with substrates, scaffold proteins, and regulatory phosphatases, further modulating its activity and specificity (orand2023revealingthemechanism pages 33-38).
6. Regulation  
   MAPK8/JNK1 is tightly regulated both by upstream kinases and by regulatory proteins that modulate its activity through post-translational modifications. Key to its activation is the dual phosphorylation of threonine and tyrosine residues within the conserved TPY motif in the activation loop; this phosphorylation is carried out synergistically by the dual-specificity kinases MKK4 and MKK7 (ansideri2018multiplestrategiestargeting pages 38-42, park2019mkk7theessential pages 1-2). Scaffold proteins such as JNK-interacting protein 1 (JIP1) further organize the components of the kinase cascade, ensuring efficient signal transduction and substrate specificity (orand2023revealingthemechanism pages 175-178, rehfeldt2020cjunnterminalkinase pages 5-7). Additionally, MAPK8/JNK1 is subject to regulation by dual-specificity phosphatases (DUSPs/MKPs), including MKP-1, MKP-7, and others, which dephosphorylate the activation loop and attenuate kinase signaling. Other post-translational modifications, for example, phosphorylation events beyond the activation loop on interacting proteins, also contribute to the fine-tuning of JNK1 activity; these modifications can influence the kinase’s interaction with substrate docking motifs and modify its subcellular localization (johnson2007thecjunkinasestressactivated pages 2-4, ha2019phosphorylationdynamicsof pages 15-16). The net result of these regulatory inputs is a tightly controlled signaling pathway that enables context-dependent activation of JNK1 in response to various extracellular stimuli such as pro-inflammatory cytokines, UV irradiation, reactive oxygen species, and other stress signals (ansideri2018multiplestrategiestargeting pages 38-42, johnson2007thecjunkinasestressactivated pages 4-5).
7. Function  
   MAPK8/JNK1 plays multifaceted roles in cellular biology by mediating the phosphorylation of a diverse array of substrates involved in transcriptional regulation, cell proliferation, cell differentiation, migration, apoptosis, and stress responses. One of its primary functions in the nucleus is to phosphorylate components of the activator protein 1 (AP-1) transcription factor complex, principally c-Jun, thereby modulating the expression of genes that govern cell survival, stress responses, and programmed cell death (ansideri2018multiplestrategiestargeting pages 38-42, barr2001thecjunnterminal pages 8-9). In addition, MAPK8 phosphorylates several other regulatory proteins such as ATF2, p53, and Yes-associated protein YAP1, contributing to the induction of apoptosis under conditions of cellular stress or DNA damage (johnson2007thecjunkinasestressactivated pages 4-5, bogoyevitch2006usesforjnk pages 18-19). In the context of cell cycle regulation, JNK1 phosphorylates the replication licensing factor CDT1, thereby inhibiting its interaction with the histone acetylase HBO1 and preventing the acetylation events required for replication initiation (ansideri2018multiplestrategiestargeting pages 38-42). Furthermore, JNK1 contributes to the regulation of autophagy by phosphorylating BCL2, leading to its dissociation from BECN1 and subsequent activation of the autophagic process (ansideri2018multiplestrategiestargeting pages 38-42). In immune cells, specifically T-cells, MAPK8 along with its close homolog MAPK9 is implicated in the differentiation of T-helper cells into the Th1 subtype, while in erythroid cells, JNK1-mediated phosphorylation of the pro-apoptotic protein BAD promotes cell survival following erythropoietin stimulation (johnson2007thecjunkinasestressactivated pages 5-6). Neuronal functions of JNK1 include the regulation of microtubule dynamics through phosphorylation of stathmin-like proteins such as STMN2, thereby controlling neurite elongation and neuronal migration, processes that are essential for proper brain development (ansideri2018multiplestrategiestargeting pages 38-42, orand2023revealingthemechanism pages 41-45). Moreover, JNK1 plays a role in circadian rhythm regulation through phosphorylation of the CLOCK-BMAL1 heterodimer, and it modulates stress responses by phosphorylating heat shock transcription factors such as HSF1 and HSF4 (ansideri2018multiplestrategiestargeting pages 35-38, rehfeldt2020cjunnterminalkinase pages 9-11). Collectively, these functions underscore the central role of MAPK8/JNK1 in integrating extracellular stress signals into appropriate cellular responses that impact gene expression, cell fate decisions, and homeostasis (cargnello2011activationandfunction pages 6-8, johnson2007thecjunkinasestressactivated pages 5-6).
8. Other Comments  
   Numerous small-molecule inhibitors of MAPK8/JNK1 have been developed for experimental and therapeutic purposes, reflecting its involvement in diseases such as cancer, neurodegeneration, metabolic disorders, and inflammation. Inhibitors such as SP600125 and CEP-1347 have been shown to block JNK activity in various preclinical studies, reducing apoptosis and tissue damage in models of ischemia/reperfusion injury and neurodegeneration (johnson2007thecjunkinasestressactivated pages 4-5, rehfeldt2020cjunnterminalkinase pages 9-11). Moreover, the ubiquitous involvement of MAPK8/JNK1 in stress and inflammatory signaling pathways has provided the rationale for targeting this kinase in conditions like Alzheimer’s disease, Parkinson’s disease, and chronic inflammatory disorders (rehfeldt2020cjunnterminalkinase pages 5-7, orand2023revealingthemechanism pages 77-81). Disease-associated genetic variants and aberrant activation of MAPK8 have been implicated in altered cellular responses, including defective apoptotic signaling and impaired cell cycle regulation, highlighting its relevance as a therapeutic target. Lastly, detailed computational studies have facilitated the design of non-ATP competitive inhibitors that exploit alternative binding modes in regions such as substrate docking sites, aiming to enhance the specificity of inhibition given the highly conserved nature of the ATP-binding pocket across the MAPK family (schnieders2012computationalinsightsfor pages 1-2). These aspects emphasize the dual promise of targeting JNK1 both for dissecting basic biological processes and for developing potential drug candidates (johnson2007thecjunkinasestressactivated pages 4-5).
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