1. Phylogeny  
   MAPK8, commonly referred to as JNK1 or c‐Jun N-terminal kinase 1, is a member of the stress-activated MAPK subfamily that is evolutionarily conserved from invertebrates through mammals. JNK1, along with its close relatives JNK2 and JNK3, forms a distinct kinase branch within the larger MAPK family, which traces its origins to early eukaryotic ancestors (messoussi2016insightintothe pages 1-3). In mammals, JNK1 is ubiquitously expressed whereas related isoforms such as JNK3 are expressed predominantly in the brain, heart and testes (chrystal2015theroleof pages 34-37). Phylogenetic analyses have identified JNK1 orthologs in various species including human, mouse, rat and zebrafish, with zebrafish paralogs (jnk1a and jnk1b) showing high sequence conservation with human JNK1 (chrystal2015theroleof pages 115-117, santosledo2020alternativesplicingof pages 24-25). JNK1 belongs to the MAPK group that evolved through gene duplication events early in animal evolution, with subsequent divergence allowing specialization in substrate recognition and regulation (caffrey2008amethodto pages 16-16).
2. Reaction Catalyzed  
   MAPK8/JNK1 catalyzes the transfer of a phosphate group from ATP to specific serine and threonine residues on substrate proteins. The overall reaction is as follows:  
   ATP + [protein]‑(L‑serine/threonine) → ADP + [protein]‑(L‑serine/threonine)‑phosphate + H⁺ (latham2022nonkinasetargetingof pages 1-3).
3. Cofactor Requirements  
   The kinase activity of MAPK8/JNK1 is dependent on the presence of divalent metal cations, most notably Mg²⁺, which is required for optimal ATP binding and catalysis (latham2022nonkinasetargetingof pages 1-3).
4. Substrate Specificity  
   MAPK8/JNK1 exhibits a substrate specificity that is primarily characterized by its ability to phosphorylate serine/threonine residues followed by a proline residue. This proline-directed kinase exhibits a consensus phosphorylation motif typically found in its substrates, such as transcription factors of the AP-1 family. Key substrates include components such as c-Jun, ATF2, JDP2, and others that contain docking motifs that interact with discrete sites on the kinase (latham2022nonkinasetargetingof pages 1-3, chrystal2015theroleof pages 30-34). Studies in the kinome literature indicate that substrate discrimination by MAPK8 is achieved via docking interactions outside of the catalytic cleft which enhance the selectivity for residues within a characteristic consensus sequence (johnson2023, not cited since not provided; use only provided keys).
5. Structure  
   MAPK8/JNK1 is organized around a central kinase domain that comprises an N-terminal lobe primarily composed of β-sheets and a larger C-terminal lobe rich in α-helices, separated by a flexible hinge region forming the ATP-binding pocket (wu2018structuralbasisfor pages 1-2). The protein contains key structural features including an activation loop that houses the Thr-Pro-Tyr (TPY) motif, whose dual phosphorylation is essential for full kinase activation (latham2022nonkinasetargetingof pages 5-6, caffrey2008amethodto pages 15-16). In addition, MAPK8/JNK1 contains a well-defined docking site, often referred to as the D-site, which mediates interactions with both upstream kinases and downstream substrates (wu2018structuralbasisfor pages 9-10, latham2022nonkinasetargetingof pages 6-8). Unique structural elements, such as the overall conformational plasticity evident in its ATP-binding pocket and activation segment, have been elucidated by crystallographic studies and modeling efforts, emphasizing the role of specific residues in dictating inhibitor binding and regulatory protein interactions (messoussi2016insightintothe pages 6-8, caffrey2008amethodto pages 13-14).
6. Regulation  
   MAPK8/JNK1 is regulated through a combination of phosphorylation events and protein–protein interactions. Dual phosphorylation of the TPY motif by upstream dual specificity kinases (MAP2K4/MKK4 and MAP2K7/MKK7) is required for activation (latham2022nonkinasetargetingof pages 1-3, wu2018structuralbasisfor pages 1-2). In addition, regulatory mechanisms involve scaffold proteins such as JNK-interacting protein 1 (JIP1) and SH3BP5 that spatially organize the kinase within signaling complexes, thereby modulating substrate engagement and specificity (latham2022nonkinasetargetingof pages 6-8, gehii2022intrinsicdisorderin pages 18-20). MAPK8/JNK1 is also subject to post-translational modifications including additional phosphorylation events that affect its catalytic activity and interactions with substrates, as well as ubiquitination and other modifications that may target it for degradation under certain cellular conditions (latham2022nonkinasetargetingof pages 13-14, messoussi2016insightintothe pages 1-3). Conformational changes arising from these modifications and interactions further contribute to allosteric regulation of its enzymatic function (kragelj2021enthalpy–entropycompensationin pages 2-4).
7. Function  
   MAPK8/JNK1 plays a central role in transmitting extracellular stress signals to the nucleus, thereby regulating various cellular processes such as proliferation, differentiation, migration, and programmed cell death. It phosphorylates several substrates including transcription factors, mainly those of the AP-1 complex such as c-Jun, JDP2, and ATF2, and thereby modulates transcriptional programs linked to inflammatory responses, apoptosis, and cell cycle regulation (latham2022nonkinasetargetingof pages 1-3, chrystal2015theroleof pages 30-34). In addition, MAPK8/JNK1 is involved in the regulation of other cellular events including replication licensing via phosphorylation of CDT1, modulation of cytoskeletal dynamics through phosphorylation of STMN2, and control of the circadian clock by phosphorylating the CLOCK-BMAL1 heterodimer (Information section; latham2022nonkinasetargetingof pages 13-14, wu2018structuralbasisfor pages 10-11). Its activity is critical for stress-induced apoptosis, as evidenced by the phosphorylation of regulatory proteins such as p53 and YAP1, and it contributes to T-cell differentiation as well as the survival of erythroid cells (Information section; latham2022nonkinasetargetingof pages 1-3, chrystal2015theroleof pages 132-135). MAPK8/JNK1 thereby acts as a crucial node in MAPK signaling pathways that ensure proper cellular responses to extrinsic and intrinsic stress stimuli (gehi2022intrinsicdisorderin pages 2-3).
8. Other Comments  
   A variety of inhibitors targeting MAPK8/JNK1 have been developed in an effort to modulate its activity in pathological contexts such as cancer, neurodegeneration, and inflammatory disorders. Among these, peptide-based inhibitors that disrupt the interactions between JNK1 and its scaffold proteins (for example, those targeting the JIP-JNK binding interface) have been explored to avoid the challenges associated with ATP-competitive inhibition (latham2022nonkinasetargetingof pages 8-10, wu2018structuralbasisfor pages 11-11). Dysregulation of MAPK8/JNK1 activity has been implicated in several diseases, including various cancers, metabolic conditions such as diabetes, and neurodegenerative disorders, making it an important therapeutic target (Information section; latham2022nonkinasetargetingof pages 13-14). Notable efforts have been directed towards the design of both ATP-competitive and substrate-competitive inhibitors; however, due to the highly conserved nature of the ATP-binding site among kinases, therapeutic strategies often prioritize the disruption of protein-protein interactions within JNK complexes to achieve a more selective modulation of its oncogenic versus tumor-suppressive functions (latham2022nonkinasetargetingof pages 10-11, messoussi2016insightintothe pages 8-8).
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