1. Phylogeny  
   Mitogen‐activated protein kinase 10 (MAPK10), more commonly known as JNK3 (also referred to as JNK3A, PRKM10, SAPK1B, or MAP kinase p49 3F12), is a member of the expansive mitogen‐activated protein kinase (MAPK) family that comprises numerous serine/threonine protein kinases involved in cellular signal transduction (al.)2002mitogenactivatedproteinkinase pages 3-3). Within this family, MAPK10 is specifically categorized in the c‐Jun N‐terminal kinase (JNK) subgroup that also includes the ubiquitously expressed isoforms JNK1 (MAPK8) and JNK2 (MAPK9); however, MAPK10 is distinguished by its predominant expression in neuronal tissues (al.)2002mitogenactivatedproteinkinase pages 6-7). Evolutionary studies, including those analyzing the protein kinase complement of the human genome, indicate that the JNK subgroup is nested within the larger CMGC group, which unites cyclin‐dependent kinases (CDKs), glycogen synthase kinases (GSKs), CDC‐like kinases (CLKs) and other kinases that share highly conserved catalytic motifs and overall sequence similarity (al.)2002mitogenactivatedproteinkinase pages 3-3). Gene duplication events early in metazoan evolution have paved the way for the diversification of the JNK family, whereby MAPK10 evolved to serve specialized roles in the nervous system; orthologs of MAPK10 have been identified across a diverse range of species from invertebrates to mammals, underscoring its evolutionary conservation and adaptation for mediating stress‐activated responses in neural contexts (krishna2008thecomplexityof pages 1-2, al.)2002mitogenactivatedproteinkinase pages 3-3).
2. Reaction Catalyzed  
   MAPK10 functions as a serine/threonine protein kinase that catalyzes the phosphorylation of target proteins by transferring the γ‐phosphate group from ATP to hydroxyl groups on serine or threonine residues. The chemical reaction can be described by the classical scheme:  
     ATP + [protein]-(L‑serine or L‑threonine) → ADP + [protein]-(phospho‑L‑serine or phospho‑L‑threonine) + H⁺ (al.)2002mitogenactivatedproteinkinase pages 3-3). This reaction is fundamental to the regulation of downstream signaling processes, as the addition of a phosphate moiety modulates the activity, stability, and subcellular localization of various substrates involved in cellular stress and apoptotic pathways.
3. Cofactor Requirements  
   The enzymatic activity of MAPK10 is critically dependent on the presence of divalent metal cations. In particular, magnesium (Mg²⁺) serves as an essential cofactor by binding to ATP in the active site, thereby stabilizing the molecule and properly orienting its γ‐phosphate for transfer to the target serine or threonine residue (coffey2014nuclearandcytosolic pages 1-2). This requirement is a hallmark of serine/threonine kinases and is shared by all members of the MAPK family.
4. Substrate Specificity  
   Experimental substrate specificity studies for human serine/threonine kinases demonstrate that MAPK10 has a marked preference for phosphorylating serine or threonine residues immediately followed by a proline residue, giving rise to the minimal consensus phosphorylation motif [S/T]P. This proline-directed specificity is a defining characteristic of the JNK subgroup and is critical for the phosphorylation of key substrates, especially transcription factors that are part of the activator protein 1 (AP‑1) complex, such as c‑Jun and ATF2 (ha2019phosphorylationdynamicsof pages 1-3). In addition, further refinement of substrate recognition is conferred by surrounding amino acids – for instance, basic residues at positions –3 and –2 relative to the phosphorylated residue can enhance substrate affinity and catalytic turnover, although the prime requisite remains the presence of the proline at the +1 position (johnson2023anatlasof pages 4-4, ha2019phosphorylationdynamicsof pages 1-3). These critical features enable MAPK10 to selectively target substrates that regulate cellular stress responses and neuronal apoptosis.
5. Structure  
   MAPK10 adopts the canonical two‐lobed structure characteristic of MAP kinases. Its structure is organized into a small N-terminal lobe that consists predominantly of β-sheets and contains a glycine-rich loop (G-loop) which confers the flexibility needed for ATP binding, and a larger C-terminal lobe that is mainly α-helical and provides the platform for substrate binding and catalysis (al.)2002mitogenactivatedproteinkinase pages 6-7). The enzyme’s central catalytic domain houses an activation loop that contains the conserved Thr–Pro–Tyr (TPY) motif; dual phosphorylation of the threonine and tyrosine residue within this loop by the upstream kinases MAP2K4 (MKK4) and MAP2K7 (MKK7) triggers a conformational rearrangement that transitions MAPK10 from an inactive to an active state (bogoyevitch2006usesforjnk pages 4-6, bogoyevitch2010cjunnterminalkinase pages 6-7). This phosphorylation realigns critical catalytic residues, stabilizes the formation of a hydrophobic spine, and optimizes the conformation of the C-helix to establish a properly configured ATP-binding pocket. Beyond its catalytic domain, MAPK10 contains several docking grooves and D-domains that facilitate high-affinity interactions with substrates, scaffold proteins, and upstream activators; these regions, although distinct from the catalytic cleft, are pivotal for ensuring signaling specificity by mediating the assembly of multiprotein signaling complexes (gordon2013combiningdockingsite pages 15-17). The overall structure, as derived from crystallographic studies and reinforced by AlphaFold models, confirms that MAPK10 shares the highly conserved structural architecture of MAP kinases while exhibiting subtle isoform-specific variations that may contribute to its neuronal selectivity.
6. Regulation  
   The regulatory mechanisms governing MAPK10 activity are multi-layered and predominantly involve post-translational modifications. The primary regulatory event is the dual phosphorylation that occurs on the activation loop’s TPY motif; phosphorylation at both the threonine and the tyrosine residues by the upstream dual-specificity kinases MAP2K4 (MKK4) and MAP2K7 (MKK7) is indispensable for full catalytic activation of MAPK10 (barr2001thecjunnterminal pages 1-3, bogoyevitch2006usesforjnk pages 1-2). This dual phosphorylation induces a significant conformational change that opens the substrate-binding cleft and aligns the catalytic machinery for efficient phosphoryl transfer. In addition to phosphorylation, MAPK10 is subject to regulation via protein–protein interactions. Scaffold proteins – notably members of the JNK-interacting protein (JIP) family – serve to tether MAPK10 with its activators and substrates, thus enhancing the specificity and efficiency of the signaling cascade (herdegen2005contextspecificinhibitionof pages 1-2, jha2025deeplearningcoupledproximity pages 10-11). Furthermore, non-degradative ubiquitination has been shown to modulate MAPK10 activity by altering its subcellular localization or interaction potential without triggering proteasomal degradation (ball2016nondegradativeubiquitinationof pages 40-41). Finally, the inactivation of MAPK10 is achieved via the action of specific phosphatases that dephosphorylate the activation loop, thereby returning the kinase to an inactive conformation in the absence of continued upstream signaling (bogoyevitch2006usesforjnk pages 2-3).
7. Function  
   MAPK10 is predominantly expressed in neuronal tissues and plays a central role in mediating various aspects of neuronal physiology. It is critically involved in the regulation of neuronal proliferation, differentiation, migration, and programmed cell death (apoptosis) (barr2001thecjunnterminal pages 1-3, coffey2014nuclearandcytosolic pages 1-2). As a key component of the stress-activated protein kinase (SAPK)/JNK signaling pathway, MAPK10 is rapidly activated in response to extracellular stress signals such as pro-inflammatory cytokines or physical stress. Once activated, MAPK10 phosphorylates a suite of downstream substrates that collectively modulate cellular responses: its phosphorylation of transcription factors such as c-Jun and ATF2 is crucial for the regulation of AP-1–dependent gene expression programs implicated in stress responses and apoptosis (bogoyevitch2006usesforjnk pages 25-26, barr2001thecjunnterminal pages 1-3). In addition, MAPK10 phosphorylates JUND – a phosphorylation event that is specifically inhibited by the MEN1 protein – further fine-tuning the transcriptional output (bogoyevitch2006usesforjnk pages 4-6). Beyond transcription factors, MAPK10 targets neuronal-specific proteins; for example, its phosphorylation of STMN2, a microtubule regulator, plays a significant role in modulating cytoskeletal dynamics necessary for neurite outgrowth and neuronal process formation (ferrer2022dysregulatedproteinphosphorylation pages 30-33). Moreover, MAPK10 phosphorylates the amyloid precursor protein (APP), thereby influencing APP-mediated signaling pathways that are critical during neuronal differentiation. Its role extends to the regulation of circadian rhythms through phosphorylation of the CLOCK-BMAL1 heterodimer, linking neuronal stress signaling to the control of daily physiological cycles (ferrer2022dysregulatedproteinphosphorylation pages 1-5, bogoyevitch2006usesforjnk pages 25-26). Collectively, these functions underscore MAPK10’s pivotal role in integrating extracellular stress signals with diverse intracellular responses that are fundamental to both neural development and the adaptive responses of neurons.
8. Other Comments  
   MAPK10’s neuronal-restricted expression and its integral role in mediating stress-activated apoptotic signaling have made it a significant focus of therapeutic research. Several experimental inhibitors targeting the JNK family act by binding to the ATP-binding site or by disrupting critical docking interactions between MAPK10 and its substrates or scaffold proteins. Although most inhibitors developed to date are pan-JNK inhibitors, current research is increasingly directed toward refining isoform selectivity so as to preferentially inhibit MAPK10 while minimizing off-target effects on JNK1 and JNK2 (latham2022nonkinasetargetingof pages 1-3, latham2022nonkinasetargetingof pages 13-14). Furthermore, dysregulation of MAPK10-mediated phosphorylation has been implicated in a number of neurodegenerative disorders, including Alzheimer’s disease and acute neuronal injury, implicating overactivation of MAPK10 in pathological neuronal apoptosis (latham2022nonkinasetargetingof pages 5-6). In addition, structure-based virtual screening and in vitro assays have identified candidate molecules with neuroprotective potential that selectively inhibit MAPK10 activity, forming the basis for ongoing drug development efforts (rajan2020identificationandneuroprotective pages 11-12). Research has also highlighted the role of non-degradative ubiquitination in fine-tuning MAPK10 activity, suggesting that modulation of these post-translational modifications may provide alternative avenues to achieve therapeutic intervention while maintaining normal neuronal function (ball2016nondegradativeubiquitinationof pages 40-41).
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