1. Phylogeny  
   MAPK10, also known as JNK3, is a member of the stress‐activated branch of the mitogen‐activated protein kinase (MAPK) family that evolved from a common ancestral serine/threonine kinase found in early eukaryotes. Unlike its paralogs JNK1 and JNK2—which are expressed ubiquitously across mammalian tissues—JNK3 exhibits a more restricted expression pattern, being expressed predominantly in neuronal tissues, as well as in the heart and testes. Comparative sequence analyses have shown that while the catalytic kinase domain of JNK3 is highly conserved with those of JNK1 and JNK2, regions outside the catalytic core have diverged and now mediate selective interactions with substrates and regulatory proteins. This divergence is consistent with phylogenetic studies demonstrating the evolution of the MAPK family from simpler kinases present in yeast to a more diversified kinome in mammals. Large-scale kinome analyses have established that the MAPK family, including the JNK isoforms, shares an evolutionary origin that can be traced back to the Last Eukaryotic Common Ancestor, with subsequent gene duplications and functional specializations leading to the tissue‐specific expression of JNK3 in the central nervous system and select peripheral tissues (kyriakis2012mammalianmapksignal pages 2-3, ansideri2018multiplestrategiestargeting pages 38-42).
2. Reaction Catalyzed  
   MAPK10/JNK3 catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of serine or threonine residues in protein substrates. In this reaction, ATP and a target protein are converted to ADP and a phosphorylated protein product, with the general chemical equation formalized as: ATP + protein-(L-serine or L-threonine) → ADP + protein-(L-serine/threonine)-phosphate + H⁺. This phosphorylation event modulates the structural conformation, activity, stability, and interaction potential of the substrate proteins, serving as a critical regulatory switch in various intracellular signaling cascades that mediate cellular stress responses and gene expression (bardwell2006mechanismsofmapk pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of MAPK10/JNK3 is dependent on the presence of divalent metal ions, with magnesium (Mg²⁺) being essential. Mg²⁺ serves to coordinate the binding of ATP within the active site of the kinase, thereby facilitating the optimal positioning of the phosphate group for its subsequent transfer to the hydroxyl group of the target serine or threonine residue. This cofactor requirement is characteristic of many serine/threonine kinases and is crucial for the enzymatic function of MAPK10/JNK3 (bardwell2006mechanismsofmapk pages 1-2).
4. Substrate Specificity  
   MAPK10/JNK3 exhibits a distinct substrate specificity that extends beyond the minimal recognition of serine or threonine residues followed by proline (S/T-P motif), a feature common to many MAPK substrates. Although the S/T-P motif is widespread, its prevalence is significantly refined in the context of JNK3 through the influence of additional substrate-docking interactions. Many substrates of JNK3 harbor specific docking sites (often referred to as D-sites), which are short linear motifs composed of a cluster of basic residues followed by hydrophobic residues. These docking regions interact with complementary grooves on the kinase surface, thereby enhancing substrate affinity and ensuring precise phosphorylation events. For instance, transcription factors such as c-Jun and ATF2—well-known targets of MAPK10/JNK3—contain structured D-sites that mediate their recruitment to the kinase, thereby ensuring that phosphorylation occurs at designated serine or threonine residues within the S/T-P context. Moreover, neuronal-specific substrates like the microtubule regulator STMN2 and the amyloid-beta precursor protein (APP) also display docking features that refine the substrate specificity of JNK3, aligning with its specialized functions in neuronal signaling and stress response (whisenant2010computationalpredictionand pages 1-2, gordon2013combiningdockingsite pages 8-10, ansideri2018multiplestrategiestargeting pages 38-42, kyriakis2012mammalianmapksignal pages 9-10).
5. Structure  
   The three-dimensional organization of MAPK10/JNK3 conforms to the canonical kinase fold observed across the MAPK family. Its structure is composed of a bilobed architecture wherein a smaller N-terminal lobe, predominantly formed by a five-stranded antiparallel β-sheet, is juxtaposed against a larger C-terminal lobe that is rich in α-helices. A key component of the N-terminal lobe is the glycine-rich loop, which is positioned between the β1 and β2 strands and plays an essential role in coordinating the phosphates of bound ATP. Adjacent to this loop is the αC-helix, a structural element critical for orienting key catalytic residues necessary for efficient phosphoryl transfer. Central to the regulation of MAPK10/JNK3 is the activation loop, within which lies a conserved threonine–proline–tyrosine (TxY) motif—specifically, Thr183-Pro-Tyr185. Dual phosphorylation of this TxY motif by upstream kinases such as MAP2K4 and MAP2K7 is required for the full activation of JNK3, driving conformational shifts that facilitate substrate access to the catalytic site while simultaneously contributing to the formation of a hydrophobic spine that stabilizes the active conformation (lu2023developmentofa pages 3-5, messoussi2016insightintothe pages 1-3, kyriakis2012mammalianmapksignal pages 8-9).  
   In addition to its highly ordered kinase domain, MAPK10/JNK3 possesses regions of intrinsic disorder, particularly in its C-terminal region. These intrinsically disordered protein regions (IDPRs) provide flexible interaction surfaces, enabling dynamic binding to an array of substrates, scaffold proteins, and regulatory factors. High-resolution structures obtained from X-ray crystallography, combined with high-confidence predictions from AlphaFold models, underscore the integrity of the catalytic core while also revealing distinct docking grooves that facilitate the binding of substrate D-sites. This dual arrangement—of a rigid catalytic core alongside flexible, disordered segments—underpins the capacity of JNK3 to integrate multiple regulatory inputs while maintaining catalytic efficacy (whisenant2010computationalpredictionand pages 1-2, giới2022intrinsicdisorderin pages 18-20, whisenant2010computationalpredictionand pages 16-17).
6. Regulation  
   Regulation of MAPK10/JNK3 is achieved through a series of tightly controlled mechanisms that are responsive to extracellular stress signals. The principal regulatory event involves the dual phosphorylation of the activation loop at the TxY motif, an event carried out by the dual-specificity kinases MAP2K4 (MKK4) and MAP2K7 (MKK7). This modification induces conformational changes in JNK3 that convert it from an inactive to an active state, thereby unleashing its catalytic potential. In addition to the modification of the activation loop, MAPK10/JNK3 undergoes regulation via specific docking interactions. Many substrates of JNK3 contain defined D-sites, and these motifs facilitate the enzyme’s interaction with substrate proteins by targeting them to specific binding grooves on the kinase surface. Such docking interactions not only refine substrate specificity but also enhance the local concentration of substrates at the active site, increasing phosphorylation efficiency (ansideri2018multiplestrategiestargeting pages 38-42, whisenant2010computationalpredictionand pages 5-7, gordon2013combiningdockingsite pages 15-17).  
   Furthermore, in neuronal cells, the activity of MAPK10/JNK3 is modulated by scaffold proteins, including JNK-interacting protein 1 (JIP1) and β-arrestin2. These scaffold proteins assemble multiprotein complexes that serve to spatially restrict JNK3, dictate its subcellular localization, and coordinate the phosphorylation of specific downstream targets. Through such interactions, the cell is able to integrate extracellular signals such as pro-inflammatory cytokines and physical stress cues, ensuring that the resultant kinase activity is appropriately contextualized. Additional regulatory inputs may also involve protein–protein interactions that can either enhance or inhibit substrate phosphorylation. For example, the interaction with MEN1 has been shown to inhibit the phosphorylation of the transcription factor JUND by JNK3, thereby modulating downstream transcriptional responses (kyriakis2012mammalianmapksignal pages 9-10, musi2022colocalizationandinteraction pages 12-13).
7. Function  
   MAPK10/JNK3 is intricately involved in mediating the cellular response to various stressors and plays a pivotal role in neuronal signaling. Because of its predominant expression in neuronal tissues, JNK3 is critically implicated in processes such as neuronal proliferation, differentiation, migration, and programmed cell death. Activation of JNK3 by extracellular stress signals—including those initiated by pro-inflammatory cytokines and physical stress—leads to the phosphorylation of a range of substrates, particularly transcription factors such as members of the AP-1 complex. Phosphorylation of transcription factors such as c-Jun and ATF2 by JNK3 alters their transcriptional activity, thereby modulating gene expression programs that govern neuronal apoptosis, inflammation, and stress responses (musi2022colocalizationandinteraction pages 10-12, kyriakis2012mammalianmapksignal pages 7-8).  
   In addition to transcription factors, JNK3 phosphorylates proteins that are important for the regulation of neural structure and function. For instance, phosphorylation of the microtubule regulator STMN2 by JNK3 is key for neurite outgrowth and axonal growth, processes that are vital both during neural development and in regenerative responses following injury. Furthermore, MAPK10/JNK3 is involved in the regulation of amyloid-beta precursor protein (APP) signaling during neuronal differentiation, a process relevant to the pathophysiology of neurodegenerative disorders. JNK3 has also been shown to target components of the circadian clock such as the CLOCK–BMAL1 heterodimer, thereby linking stress signaling with the modulation of circadian rhythms. These diverse roles underscore the importance of MAPK10/JNK3 in integrating extracellular stress cues with intracellular signaling networks, ultimately affecting both immediate cellular responses and long-term regulatory processes in neurons (ansideri2018multiplestrategiestargeting pages 38-42, kyriakis2012mammalianmapksignal pages 7-8, lu2023developmentofa pages 3-5).
8. Other Comments  
   MAPK10/JNK3 has emerged as a key target for therapeutic intervention, especially in neurodegenerative diseases such as Alzheimer’s disease where aberrant activation of stress-induced apoptotic pathways may contribute to pathological neuronal loss. This has driven significant efforts toward the development of small-molecule inhibitors that target JNK3 selectively, with particular emphasis on sparing JNK1, which is more widely expressed. Advanced structure-based drug design approaches have been employed to develop covalent inhibitors that preferentially target JNK3 as well as JNK2 by exploiting subtle differences in the ATP-binding pocket and regulatory regions; these efforts are illustrated in studies that approach the structural promiscuity and ligand docking conformations of JNK isoforms (lu2023developmentofa pages 3-5, sailapathi2020proposingthepromiscuous pages 9-10).  
   Moreover, the regulation of MAPK10/JNK3 by scaffold proteins such as JIP1 and β-arrestin2 has also stimulated interest in targeting protein–protein interactions as a means to modulate kinase activity. These scaffolding interactions are critical for determining the subcellular distribution and temporal activation of JNK3, and thereby its downstream effects on apoptosis, neurite outgrowth, and synaptic plasticity. Computational tools, including the D-finder algorithm, have further expanded the known substrate repertoire of JNK3 by identifying novel docking sites that enhance substrate specificity. Such integrative approaches promise to refine our understanding of MAPK10/JNK3 signaling networks and to inform the development of next-generation therapeutics that precisely target JNK3-mediated pathways without off-target effects on related kinases (whisenant2010computationalpredictionand pages 7-9, gordon2013combiningdockingsite pages 15-17).
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