1. Phylogeny  
   MAP3K10, also known as MLK2 or MST, belongs to the serine/threonine kinase superfamily and is classified specifically as a MAP kinase kinase kinase (MAP3K) within the mixed lineage kinase (MLK) subgroup of the tyrosine kinase‐like (TKL) family. Its evolutionary origin can be traced to the ancient protein kinase network conserved from yeast to man, with orthologs identified in all vertebrate lineages, thereby underscoring its pivotal role in intracellular signal transduction (cargnello2011activationandfunction pages 5-6, li2011evolutionaryhistoryof pages 11-12). Phylogenetic analyses have demonstrated that MAP3K10 is positioned alongside other MAP3Ks that participate in stress‐activated signaling cascades, and its orthologs share a high degree of conservation in their kinase catalytic domains across diverse mammalian species (gonzalezcoronel2021aphylogeneticstudy pages 15-17, li2011evolutionaryhistoryof pages 11-12). In the context of the human kinome, MAP3K10 represents a member of the MLK family that diversified alongside other mixed lineage kinases during early vertebrate evolution, a divergence that has contributed to the specialization of MAPK signaling pathways involved in stress responses (barr2001thecjunnterminal pages 3-5, cargnello2011activationandfunction pages 5-6). Its overall phylogenetic placement within the TKL group implies a shared ancestry with several other MAP3K family members that function upstream of mitogen‐activated protein kinase (MAPK) cascades, further highlighting its role as a conserved regulatory node in eukaryotic signaling (krupa2002therepertoireof pages 4-5, lehtishiu2012diversityclassificationand pages 10-11).
2. Reaction Catalyzed  
   MAP3K10 catalyzes the transfer of the γ‐phosphate group from ATP to specific serine or threonine residues on substrate proteins, a reaction fundamental to its role as a kinase in the MAPK cascade. In biochemical terms, the enzymatic reaction can be summarized as follows: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺, an outcome that represents the phosphorylation of substrates and the concomitant production of ADP (cargnello2011activationandfunction pages 1-2, jiang2022mitogenactivatedproteinkinase pages 2-4). This phosphorylation event is critical for the activation of downstream MAP kinase kinases (MAP2Ks), which in turn relay the signal to MAP kinases such as JNK, thereby propagating a cellular response to external stimuli (jiang2022mitogenactivatedproteinkinase pages 2-4, barr2001thecjunnterminal pages 3-5). The reaction follows the canonical kinetics of serine/threonine kinases, whereby substrate binding, ATP docking, and the proper orientation of the catalytic residues within the active site facilitate efficient phosphoryl transfer (cargnello2011activationandfunction pages 1-2). This overall phosphorylation mechanism is a defining feature of MAP3K10’s role within the hierarchical kinase cascades that regulate stress responses and apoptotic signaling (jiang2022mitogenactivatedproteinkinase pages 2-4, barr2001thecjunnterminal pages 3-5).
3. Cofactor Requirements  
   The catalytic activity of MAP3K10 is dependent upon the presence of divalent metal ion cofactors, with Mg²⁺ being the primary ion required to mediate the phosphotransfer reaction. Mg²⁺ coordinates with the ATP molecule within the active site of the kinase, thereby stabilizing the negative charges on the phosphate groups and facilitating their transfer to the substrate (cargnello2011activationandfunction pages 5-6, jiang2022mitogenactivatedproteinkinase pages 2-4). This requirement for Mg²⁺ is consistent with the general biochemical properties of serine/threonine kinases, and its presence is essential for the structural organization of the active site as well as for achieving the catalytic transition state (cargnello2011activationandfunction pages 5-6). The precise coordination of Mg²⁺ within the kinase domain not only aids in ATP binding but also plays a pivotal role in the stabilization of the phosphorylated intermediate during the reaction cycle (jiang2022mitogenactivatedproteinkinase pages 2-4).
4. Substrate Specificity  
   MAP3K10 exhibits substrate specificity that is primarily directed toward the activation loops of MAP kinase kinases (MAP2Ks), such as MKK4 and MKK7, which are integral components of the JNK signaling pathway. The kinase preferentially phosphorylates serine and threonine residues located within the dual phosphorylation sites of these MAP2Ks, a modification necessary for their subsequent activation (barr2001thecjunnterminal pages 3-5, cargnello2011activationandfunction pages 5-6). Although a detailed consensus phosphorylation motif specific to MAP3K10 has not been fully delineated in the literature, substrate profiling using peptide-based arrays suggests that related serine/threonine kinases favor sequences that feature basic residues—often arginine at the –3 position—and a modest preference for a proline residue immediately following the phosphorylatable serine/threonine (johnson2023anatlasof pages 4-5, jiang2022mitogenactivatedproteinkinase pages 9-10). This substrate preference is consistent with the overall mechanism in which MAP3K10 phosphorylates activation loops on MAP2Ks, thereby establishing a critical link in the sequential phosphorylation cascade that ultimately results in the activation of MAPKs such as JNK (zeke2015systematicdiscoveryof pages 1-3, jiang2022mitogenactivatedproteinkinase pages 9-10). Thus, while the precise consensus motif is not exclusively defined for MAP3K10, the enzyme’s substrate specificity aligns with the conserved features observed among kinases that regulate stress-activated signaling pathways.
5. Structure  
   The structural organization of MAP3K10 is characterized by a central catalytic kinase domain that encompasses the 11 conserved subdomains typical of serine/threonine protein kinases. This catalytic domain includes critical elements such as the ATP-binding pocket, the catalytic loop, the DFG motif, and the activation loop, which is subject to phosphorylation and subsequent conformational rearrangement (barr2001thecjunnterminal pages 3-5, li2011evolutionaryhistoryof pages 11-12). In addition to the central kinase domain, MAP3K10 is reported to possess regulatory modules that may include leucine/isoleucine zipper motifs, SH3 domains, and putative CRIB motifs; these domains facilitate homodimerization and interactions with small GTPases or adaptor proteins, thus modulating its activity (krupa2002therepertoireof pages 4-5, barr2001thecjunnterminal pages 3-5). Although high-resolution crystallographic data for MAP3K10 itself are limited, structural insights extrapolated from related MLK family members indicate that the 3D conformation of the kinase domain features a well-defined C-helix responsible for proper alignment of catalytic residues and a hydrophobic spine that supports the active conformation during phosphoryl transfer (li2011evolutionaryhistoryof pages 11-12, krupa2002therepertoireof pages 4-5). Computational models, including those derived from AlphaFold, further corroborate the presence of these key structural elements, and they suggest that flexible, intrinsically disordered regions flanking the catalytic core may serve regulatory functions by mediating dynamic protein–protein interactions (barr2001thecjunnterminal pages 3-5, lehtishiu2012diversityclassificationand pages 10-11). The activation loop itself is a critical regulatory segment whose phosphorylation is required to shift MAP3K10 from an autoinhibited state to an active form, thereby enabling efficient substrate phosphorylation (barr2001thecjunnterminal pages 12-14, krupa2002therepertoireof pages 4-5).
6. Regulation  
   Regulation of MAP3K10 is achieved primarily through phosphorylation of residues located within its activation loop, leading to conformational changes that relieve autoinhibition and enhance enzymatic activity. Both autophosphorylation and phosphorylation by upstream kinases contribute to its activation, and these modifications are essential for transmitting extracellular stress signals to downstream MAP kinase cascades (barr2001thecjunnterminal pages 12-14, cargnello2011activationandfunction pages 5-6). In various experimental systems, inhibitors such as CEP-1347 have been employed to target the MLK family, resulting in the suppression of MAP3K10-mediated activation of JNK pathways; such inhibitors serve as pharmacological tools to interrogate the role of MAP3K10 in stress and apoptotic signaling (barr2001thecjunnterminal pages 12-14, cargnello2011activationandfunction pages 5-6). Additionally, interactions with small GTPases and adaptor proteins are believed to further regulate MAP3K10 by facilitating its proper subcellular localization and substrate recruitment, although the precise mechanisms of these interactions remain less clearly defined in the available literature (cargnello2011activationandfunction pages 5-6, krupa2002therepertoireof pages 4-5). Collectively, these regulatory mechanisms ensure that MAP3K10 activity is tightly controlled in response to extracellular cues, thereby maintaining the fidelity of downstream signaling events within the JNK cascade (barr2001thecjunnterminal pages 12-14, cargnello2011activationandfunction pages 5-6).
7. Function  
   MAP3K10 plays a central role in the activation of the JUN N-terminal kinase (JNK) pathway, a critical signaling route involved in cellular responses to stress, inflammation, and apoptotic stimuli. By phosphorylating key MAP kinase kinases (MAP2Ks) such as MKK4 and MKK7, MAP3K10 initiates a phosphorylation cascade that culminates in the activation of JNK, which in turn modulates the activity of transcription factors including c-Jun (cargnello2011activationandfunction pages 1-2, barr2001thecjunnterminal pages 3-5). The activation of JNK by MAP3K10 contributes to the regulation of diverse biological processes encompassing programmed cell death, differentiation, and cellular adaptation to environmental stressors, such as pro-inflammatory cytokines and ultraviolet radiation (jiang2022mitogenactivatedproteinkinase pages 16-17, barr2001thecjunnterminal pages 3-5). MAP3K10 is expressed in multiple tissue types, and its activity is integral to the orchestration of stress-activated responses that balance survival and apoptosis, which is especially pertinent in neuronal cells where regulation of JNK signaling has been linked to neurodegenerative disorders (cargnello2011activationandfunction pages 1-2, jiang2022mitogenactivatedproteinkinase pages 16-17). As a key regulatory node, MAP3K10 not only mediates signal amplification within the MAPK cascade but also serves as an interface through which extracellular cues are integrated into precise transcriptional outcomes, thereby influencing cellular fate decisions (barr2001thecjunnterminal pages 3-5, jiang2022mitogenactivatedproteinkinase pages 16-17).
8. Other Comments  
   Inhibitors targeting the MLK family, including MAP3K10, have been actively investigated for their potential to mitigate neurodegenerative conditions by attenuating stress-induced JNK activation; one such inhibitor, CEP-1347, has demonstrated the ability to suppress MAP3K10 activity in preclinical models (barr2001thecjunnterminal pages 12-14, johnson2023anatlasof pages 1-2). Although detailed inhibitor profiles specific to MAP3K10 are not extensively reported, the pharmacological modulation of MAP3K10 is recognized as a promising strategy for therapeutic intervention in pathologies where aberrant stress signaling and apoptosis contribute to disease progression (johnson2023anatlasof pages 4-5, zeke2015systematicdiscoveryof pages 19-20). MAP3K10 is also known by its alternative designations, Mixed Lineage Kinase 2 and MST, which reflect its structural characteristics and functional role within the broader MAPK signaling network (barr2001thecjunnterminal pages 3-5). Dysregulation of MAP3K10-mediated signaling is associated with pathological conditions that involve excessive or insufficient activation of the JNK pathway, thereby implicating this kinase in disorders related to inflammatory responses and cellular stress (li2011evolutionaryhistoryof pages 11-12, johnson2023anatlasof pages 4-5). While specific disease mutations within the MAP3K10 gene have not been conclusively reported in the referenced studies, the centrality of MAP3K10 in controlling downstream signaling cascades underscores its potential as a target for drug development aimed at restoring balanced cellular responses in affected tissues (johnson2023anatlasof pages 1-2, zeke2015systematicdiscoveryof pages 20-21).
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