1. Phylogeny  
   MAP3K5, more commonly known as Apoptosis Signal‐Regulating Kinase 1 (ASK1), is a member of the mitogen‐activated protein kinase kinase kinase (MAP3K) family that is broadly conserved among eukaryotic organisms, spanning from yeast to mammals (kyriakis2001mammalianmitogenactivatedprotein pages 44-45, horton2011themitogenactivatedprotein pages 8-9). Sequence comparisons of the kinase domain reveal strong conservation in amino acid residues essential for catalysis and substrate recognition, which situates ASK1 as part of an evolutionary core set of MAP kinase signaling components. Orthologs of ASK1 have been identified in a wide variety of species, and within mammals, paralogues such as ASK2 have been characterized; these kinases not only share high sequence similarity but also interact functionally in heteromeric complexes to regulate apoptosis and stress signaling (kyriakis2001mammalianmitogenactivatedprotein pages 44-45, obsil2017structuralaspectsof pages 1-5). Phylogenetic analyses indicate that the evolutionary origin of ASK1 predates the divergence of major eukaryotic lineages; its kinase module is thought to have arisen before the split between metazoans and fungi, consistent with the conservation observed among the MAP kinase cascades, and further supported by the overall domain organization that mirrors that of other stress‐responsive MAP3Ks (kyriakis2001mammalianmitogenactivatedprotein pages 44-45, obsilova2021structuralinsightssupport pages 1-3). In addition, the presence of related MAP3Ks, such as ASK2 and ASK3, in higher eukaryotes underscores the modular evolution of this kinase family, while the retention of key regulatory motifs further indicates the critical cellular functions these enzymes maintain.
2. Reaction Catalyzed  
   ASK1 catalyzes the transfer of a phosphate group from ATP to specific serine/threonine residues on its substrate proteins, a reaction that is central to the propagation of stress signals through MAP kinase cascades (agrahari2022crystallographicminingof pages 18-19, kyriakis2001mammalianmitogenactivatedprotein pages 44-45). In doing so, ASK1 converts ATP into ADP and a phosphorylated substrate while releasing a proton, following the general reaction mechanism common to serine/threonine kinases:  
     ATP + Protein-(L-serine/threonine) → ADP + Protein-(L-serine/threonine)-phosphate + H⁺ (agrahari2022crystallographicminingof pages 18-19, kyriakis2001mammalianmitogenactivatedprotein pages 44-45).
3. Cofactor Requirements  
   The catalytic activity of ASK1 depends on the presence of ATP as the phosphate donor and typically requires a divalent metal ion cofactor, most commonly Mg²⁺, which facilitates proper coordination of the nucleotide in the active site (win2024mitochondrialpjnktarget pages 12-12, obsil2017structuralaspectsof pages 1-5). Mg²⁺ ions stabilize the negative charges on ATP’s phosphate groups and are essential for enabling the transfer of the γ-phosphate to the acceptor hydroxyl group of serine or threonine residues on target substrates (win2024mitochondrialpjnktarget pages 12-12).
4. Substrate Specificity  
   ASK1 displays a preference for phosphorylating a set of downstream mitogen-activated protein kinase kinases (MAP2Ks), including MAP2K4 (also known as SEK1), MAP2K7, and to a lesser extent MAP2K3 and MAP2K6, which are subsequently responsible for the activation of the JNK and p38 MAPK pathways (agrahari2022crystallographicminingof pages 2-3, kyriakis2001mammalianmitogenactivatedprotein pages 44-45). The substrate recognition is largely mediated by docking interactions facilitated by regions adjacent to the catalytic domain and by specific sequence motifs in the substrate proteins; however, a defined consensus motif has not been as firmly established for ASK1 as for some other kinases (agrahari2022crystallographicminingof pages 2-3, obsilova2021structuralinsightssupport pages 3-4). In general, the substrates of ASK1 are characterized by serine/threonine residues that become phosphorylated upon binding to the kinase’s active site, thereby propagating the stress signal through the MAPK cascade (kyriakis2001mammalianmitogenactivatedprotein pages 44-45).
5. Structure  
   MAP3K5/ASK1 is a large multidomain protein comprising approximately 1374 amino acids with a modular architecture that underpins its regulatory capacity and catalytic function. The N-terminal region features a thioredoxin-binding domain (TBD), which adopts a thioredoxin-like fold characterized by a six-stranded β-sheet flanked by several α-helices; this domain mediates binding to regulatory proteins such as thioredoxin-1 (TRX1) that control the kinase’s activity under non-stress conditions (agrahari2022crystallographicminingof pages 19-20, obsil2017structuralaspectsof pages 10-13). Immediately downstream, a central regulatory region (CRR) contains multiple tetratricopeptide repeats (TPRs) and a pleckstrin homology (PH) domain that facilitate protein-protein interactions with adaptor proteins—including members of the tumor necrosis factor receptor-associated factor (TRAF) family—that modulate ASK1 oligomerization and signaling (agrahari2022crystallographicminingof pages 2-3, obsil2017structuralaspectsof pages 5-7). The core of ASK1 is its catalytic kinase domain, which exhibits the canonical bilobal structure typical of serine/threonine kinases; the smaller N-terminal lobe is predominantly composed of β-sheets, while the larger C-terminal lobe is mainly α-helical (agrahari2022crystallographicminingof pages 6-8, obsilova2021structuralinsightssupport pages 10-12). A key feature within this domain is the activation loop, which contains threonine 838—phosphorylation of which is critical for full catalytic activation—as well as conserved structural motifs such as the C-helix and a hydrophobic spine that stabilize the active conformation of the kinase (agrahari2022crystallographicminingof pages 6-8, obsilova2021structuralinsightssupport pages 12-13). C-terminal to the kinase domain, a coiled-coil (CC) region and a sterile alpha motif (SAM) domain are present; these domains are implicated in the oligomerization of ASK1, a process essential for its autophosphorylation and activation (obsil2017structuralaspectsof pages 7-10, obsilova2021structuralinsightssupport pages 13-15). Notably, the overall three-dimensional organization of ASK1 is characterized by an asymmetric homodimer arrangement in which contacts between the N-terminal regulatory domains of one protomer and the kinase domain of the other are vital for proper allosteric regulation (agrahari2022crystallographicminingof pages 13-15, obsilova2021structuralinsightssupport pages 15-16). Furthermore, redox‐sensitive cysteine residues, such as Cys250, are embedded within the TBD of ASK1, and their oxidation state directly influences binding to TRX1 and thus the kinase’s activation state (agrahari2022crystallographicminingof pages 19-20, obsilova2021structuralinsightssupport pages 4-6).
6. Regulation  
   ASK1 activity is controlled by a complex network of regulatory mechanisms that integrate multiple post‐translational modifications and protein–protein interactions. Under basal, non‐stressed conditions, ASK1 is maintained in an inactive state predominantly through its interaction with reduced thioredoxin (TRX1), which binds to the N-terminal thioredoxin‐binding domain and sterically hinders activation; oxidative stress results in the oxidation of TRX1, leading to its dissociation and subsequent activation of ASK1 (agrahari2022crystallographicminingof pages 6-8, lee2013thioredoxinandthioredoxin pages 11-12). In addition to redox regulation, phosphorylation events play a critical role in modulating ASK1 function. Autophosphorylation of threonine 838 within the activation loop is essential for catalytic activity, while inhibitory phosphorylation at residues such as serine 83, mediated by kinases like Akt, serves to suppress ASK1 activity under conditions that favor cell survival (chen2016crosstalkbetweenarg pages 1-2, lee2013thioredoxinandthioredoxin pages 12-14). ASK1 is also subject to regulation by 14-3-3 proteins; these adaptor proteins bind to phosphorylated serine residues in the C-terminal region—most notably serine 966—thereby stabilizing an inactive conformation and preventing inappropriate kinase activation (obsilova2021structuralinsightssupport pages 13-15, obsilova2021structuralinsightssupport pages 7-9). Ubiquitination constitutes another important regulatory mechanism; E3 ubiquitin ligases, such as Roquin-2, target ASK1 for polyubiquitination, resulting in its proteasomal degradation, whereas deubiquitinating enzymes like USP9X can remove ubiquitin chains to stabilize the kinase and prolong its signaling output (agrahari2022crystallographicminingof pages 11-11, agrahari2022crystallographicminingof pages 15-16). Furthermore, regulatory interactions with TRAF proteins—particularly TRAF2 and TRAF6—promote ASK1 oligomerization and activation by serving as scaffolds that facilitate trans-autophosphorylation (agrahari2022crystallographicminingof pages 2-3, kyriakis2001mammalianmitogenactivatedprotein pages 44-45). Additional layers of regulation include S-nitrosylation and arginine methylation; for instance, arginine methylation by PRMT5 at specific residues has been shown to influence subsequent inhibitory serine phosphorylation events, thereby fine-tuning ASK1 activity in response to oxidative cues (chen2016crosstalkbetweenarg pages 2-3, lee2013thioredoxinandthioredoxin pages 14-15). Collectively, these regulatory processes ensure that ASK1 activation is tightly restricted to conditions of cellular stress and that its signaling output is appropriately balanced to mediate either apoptotic or survival outcomes.
7. Function  
   ASK1 plays a central role in transducing stress signals into specific cellular responses, particularly those related to apoptosis, inflammation, and differentiation. Upon activation by various stressors – including oxidative stress, pro-inflammatory cytokines such as tumor necrosis factor (TNF) and lipopolysaccharide (LPS), and endoplasmic reticulum (ER) stress – ASK1 phosphorylates downstream MAP2Ks such as MAP2K4 (SEK1), MAP2K7, MAP2K3, and MAP2K6, thereby triggering the activation of the JNK and p38 MAPK pathways (agrahari2022crystallographicminingof pages 2-3, kyriakis2001mammalianmitogenactivatedprotein pages 44-45). Activation of these cascades leads to the transcription of pro-apoptotic genes and the activation of effector caspases, playing an essential role in mitochondria-dependent apoptosis (agrahari2022crystallographicminingof pages 20-21, obsilova2021structuralinsightssupport pages 9-10). In addition to its pro-apoptotic function, ASK1 contributes to the regulation of the innate immune response, where it is required for the cellular defense against pathogens by modulating signals downstream of pattern recognition receptors; this function is particularly critical in hepatocytes and immune cells where ASK1-driven signaling is part of the host defense mechanism (agrahari2022crystallographicminingof pages 2-3, obsilova2021structuralinsightssupport pages 1-3). ASK1 is also implicated in processes such as cellular differentiation and tissue remodeling, with its activity influencing both the survival and the fate of cells in response to environmental cues – a balance that is crucial for maintaining homeostasis under stress conditions (agrahari2022crystallographicminingof pages 6-8, obsilova2021structuralinsightssupport pages 15-16). Notably, pathological dysregulation of ASK1 has been associated with a number of diseases, including cardiovascular disorders, neurodegenerative diseases, and certain cancers, owing to its critical role in mediating apoptosis and inflammatory responses (kyriakis2001mammalianmitogenactivatedprotein pages 44-45, uppugunduri2022insilicoandinvitroinvestigations pages 3-4). The expression of ASK1 is widespread, and its activity is finely tuned by both upstream signals and feedback mechanisms, which collectively determine cell fate decisions in a context-dependent manner.
8. Other Comments  
   Due to its central role in the regulation of apoptosis and stress‐induced signaling, ASK1 has been the focus of extensive therapeutic investigations. Several small-molecule inhibitors have been developed targeting ASK1, with compounds such as selonsertib undergoing clinical trials for conditions including nonalcoholic steatohepatitis (NASH) and cardiovascular diseases; however, complete catalytic inhibition of ASK1 has proven challenging as it may also interfere with its physiological roles in normal cell homeostasis (agrahari2022crystallographicminingof pages 2-3, obsilova2021structuralinsightssupport pages 15-16). In light of these challenges, recent strategies have shifted towards modulating protein–protein interactions that regulate ASK1 activity rather than targeting the kinase domain directly; interventions aimed at stabilizing the interaction between ASK1 and its inhibitory partners, such as TRX1 or 14-3-3, offer an alternative approach that might yield selective modulation with fewer off-target effects (agrahari2022crystallographicminingof pages 13-15, obsilova2021structuralinsightssupport pages 15-16). Furthermore, alterations in key regulatory residues—such as mutations affecting threonine 838 in the activation loop or cysteine residues critical for TRX1 binding—have been linked to aberrant ASK1 activity and may serve as potential biomarkers for diseases characterized by dysregulated apoptosis and inflammation (agrahari2022crystallographicminingof pages 18-19, chen2016crosstalkbetweenarg pages 6-7). Ongoing research continues to elucidate the structural basis of ASK1 regulation using advanced techniques such as crystallography and in silico modeling, which inform the design of next-generation inhibitors capable of precisely modulating ASK1 function in pathological states (obsil2017structuralaspectsof pages 13-14, uppugunduri2022insilicoandinvitroinvestigations pages 3-4). Given its pivotal position upstream of multiple signaling cascades, ASK1 represents a promising therapeutic target not only for traditional indications such as cardiovascular and liver diseases, but also for emerging indications in neurodegeneration and certain inflammatory conditions (win2024mitochondrialpjnktarget pages 12-12, rauch2011thesecretlife pages 11-12).

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