1. Phylogeny  
   STK35, also known as CLIK1, is a member of the Hanks‐type serine/threonine protein kinase superfamily. Sequence analyses, as demonstrated in molecular cloning experiments, indicate that STK35 shares roughly 69% amino acid identity with its paralog PDIK1L, placing these kinases together in a distinct subgroup within the CMGC branch of the kinome (guo2003molecularcloningand pages 3-6, spiridonov2005identificationandcharacterization pages 1-2). The CMGC group, comprising cyclin-dependent kinases (CDKs), mitogen-activated protein kinases (MAPKs), glycogen synthase kinases (GSKs) and related kinases, encompasses many eukaryotic representatives with conserved catalytic motifs that date back to the common ancestor of eukaryotes. Studies in organisms as diverse as Neurospora crassa have identified a homolog of stk-35 within the CLK/SRPK family of the CMGC clade, reinforcing the concept that STK35 carries evolutionarily conserved structural and functional features found throughout eukaryotic lineages (park2011globalanalysisof pages 1-2, park2011globalanalysisof pages 2-3). Moreover, analyses of the catalytic domain—which includes the glycine-rich loop (or P-loop) for ATP binding and critical motifs such as the DFG sequence at the onset of the activation segment—confirm that the evolutionary history of STK35 is marked by the preservation of key residues necessary for phosphotransfer. This conservation, observed in both human and fungal kinases, supports the notion that the fundamental biochemical activities of STK35 have been maintained since early eukaryotic evolution (guo2003molecularcloningand pages 3-6, spiridonov2005identificationandcharacterization pages 1-2). In addition, data from large-scale kinase atlases, such as those assembled through comparative sequence clustering and structural constraint analyses, indicate that STK35’s domain architecture and sequence signatures place it securely within the CMGC clade, along with other kinases known for their roles in nuclear signaling and cell cycle regulation (o’boyle2025anatlasof pages 27-31). Together, these findings underscore that STK35 is an evolutionarily ancient kinase with homologs spanning various eukaryotic taxa, maintaining a core catalytic panel that typifies the serine/threonine kinase family.
2. Reaction Catalyzed  
   STK35 catalyzes the phosphoryl transfer from ATP to specific serine or threonine residues on substrate proteins. The reaction follows the canonical mechanism observed for serine/threonine kinases and can be represented as:  
     ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(phospho-L-serine or phospho-L-threonine) + H⁺  
   This chemical transformation involves the binding of ATP in the active site of the kinase, where the γ-phosphate is positioned for transfer to the hydroxyl group of the target amino acid, resulting in the generation of a phosphorylated protein substrate and the release of ADP and a proton (spiridonov2005identificationandcharacterization pages 4-5, guo2003molecularcloningand pages 3-6). This reaction is central to the regulation of protein function and modulates downstream processes such as chromatin remodeling, gene expression, and cell cycle progression by altering substrate conformation and interaction potential without changing the polypeptide primary structure.
3. Cofactor Requirements  
   The catalytic activity of STK35 is dependent on divalent metal ions that are essential for ATP coordination and subsequent phosphoryl transfer. Consistent with its classification among serine/threonine protein kinases, STK35 requires magnesium ions (Mg²⁺) as a cofactor. Mg²⁺ ions bind to ATP and help neutralize the negative charges of the phosphate groups, thereby facilitating proper substrate orientation within the kinase active site and enhancing the efficiency of phosphoryl transfer (guo2003molecularcloningand pages 3-6, spiridonov2005identificationandcharacterization pages 4-5). Although some kinases can utilize alternative divalent cations such as Mn²⁺ to support catalytic activity, available evidence indicates that Mg²⁺ is the principal cofactor under physiological conditions for STK35.
4. Substrate Specificity  
   Experimental investigations have demonstrated that STK35 exhibits substrate specificity reflective of its nuclear functions. Biochemical assays in murine models, where STK35 (also designated as SSTK in some studies) was characterized, have identified histone proteins—such as H1, H2A, H2AX, and H3—as substrates, indicating that STK35 plays a role in modulating chromatin structure (spiridonov2005identificationandcharacterization pages 1-2, spiridonov2005identificationandcharacterization pages 4-5). In addition, general insights gleaned from studies of substrate recognition in serine/threonine kinases suggest that substrate docking is mediated by a P+1 specificity pocket. This pocket typically confers a preference for hydrophobic or basic amino acid residues immediately following the phosphorylated serine or threonine, which helps in aligning the substrate for efficient catalysis (goldsmith2007substrateanddocking pages 5-6, goldsmith2007substrateanddocking pages 6-7). Although the precise consensus phosphorylation motif for STK35 has not been completely delineated through systematic peptide library screens, it is likely that protein substrates targeted by STK35 possess specific sequence determinants near the phosphorylation site that facilitate their recognition and alignment with the catalytic cleft. This substrate specificity is critical for directing STK35’s regulatory influence on key nuclear processes, including chromatin condensation and transcriptional regulation.
5. Structure  
   High-resolution structural data for STK35 are not yet available from crystallographic studies; however, its three-dimensional organization has been inferred from sequence analyses, domain prediction algorithms, and comparisons with closely related kinases. STK35 is predicted to adopt the canonical bilobal structure common to Hanks‐type serine/threonine kinases. In this arrangement, a smaller N-terminal lobe (N-lobe) is primarily responsible for binding ATP via a glycine-rich P-loop, while a larger C-terminal lobe (C-lobe) houses the substrate-binding pocket and catalytic machinery (guo2003molecularcloningand pages 3-6, spiridonov2005identificationandcharacterization pages 4-5). The kinase domain incorporates several conserved motifs: the P-loop, the catalytic loop with an invariant lysine crucial for orienting ATP, the DFG motif that marks the beginning of the activation segment, and the APE motif towards the end of the activation loop. Such motifs are essential in aligning substrates and catalyzing phosphoryl transfer.  
   In addition, molecular cloning studies have demonstrated that constructs corresponding to the closely related paralog PDIK1L—which shares significant sequence homology with STK35—exhibit nuclear localization, implying the presence of one or more nuclear localization signals (NLSs) within non-catalytic regions of the protein (guo2003molecularcloningand pages 3-6). While the core catalytic domain is well conserved, the regions outside this domain, which may be intrinsically disordered, often serve as platforms for dynamic protein–protein interactions that modulate kinase activity. Prediction models based on established kinase structures suggest that STK35’s activation segment likely plays a key regulatory role by adopting conformational states that either permit or restrict substrate access. Comparative analyses with kinases such as those described in studies on STK16 and other CMGC kinases further support the notion that regulatory control via activation loop phosphorylation or autophosphorylation is a critical feature (wang2019serinethreonineproteinkinase pages 3-5, wang2019serinethreonineproteinkinase pages 5-7). Furthermore, insights from bacterial kinase atlases indicate that although the overall fold is conserved, subtle variations in the hydrophobic spine and the orientation of the C-helix can account for differences in substrate specificity and activity among related kinases (o’boyle2025anatlasof pages 27-31). Despite the absence of a solved crystal structure, the integration of these computational predictions provides a robust framework for understanding the domain organization and functional topology of STK35.
6. Regulation  
   Regulation of STK35 is achieved through a combination of intrinsic catalytic mechanisms and extrinsic post-translational modifications, as well as protein–protein interactions with chaperones and other regulatory factors. Mutagenesis studies have established that key residues, such as the conserved lysine found within the ATP-binding P-loop and an essential aspartate in the catalytic loop, are vital for kinase activity; their mutation leads to a complete loss of function (spiridonov2005identificationandcharacterization pages 4-5). In addition to these intrinsic determinants, STK35 is subject to reversible post-translational modifications that modulate its activity. For example, phosphoproteomic analyses have revealed modification sites on STK35, namely phosphorylation events at serine residues S941 and S37, which have been detected in studies focusing on the interplay between phosphorylation and O-GlcNAc modifications (schwein2020theoglcnacmodification pages 27-28). Although the full network of autophosphorylation sites has yet to be mapped, available evidence suggests that modifications within the activation loop may fine-tune the kinase’s catalytic cycle by influencing the conformational equilibrium between active and inactive states.  
   Further regulatory control is exerted by chaperone proteins; co-immunoprecipitation experiments have indicated that STK35 forms complexes with molecular chaperones such as HSP90-1, HSC70, and HSP70. These interactions are thought to ensure proper folding and stabilization of the kinase, especially under conditions of cellular stress, thereby preserving its functional integrity within the nucleus (spiridonov2005identificationandcharacterization pages 1-2). Moreover, the interplay between autophosphorylation and dephosphorylation events, mediated by specific phosphatases, adds an additional layer of regulation by dynamically modulating the phosphorylation status of key regulatory residues. Taken together, these regulatory mechanisms exemplify the multifaceted control that governs STK35 activity, ensuring that its function is tightly integrated with the needs of nuclear signaling and chromatin remodeling.
7. Function  
   STK35 plays pivotal roles in the regulation of nuclear processes and chromatin dynamics. Functional studies conducted in murine systems have demonstrated that genetic inactivation of STK35, sometimes referred to under the designation SSTK in early studies, leads to male sterility. This phenotype is characterized by defective spermiogenesis, which includes abnormal sperm morphology, reduced motility, and impaired chromatin condensation (spiridonov2005identificationandcharacterization pages 1-2, spiridonov2005identificationandcharacterization pages 4-5). These findings highlight a critical function for STK35 in germ cell development, where the proper phosphorylation of nuclear substrates, such as histones, is required for chromatin remodeling and the regulation of gene expression programs essential for normal sperm formation.  
   Beyond its role in the male reproductive system, expression analyses of the paralog PDIK1L—closely related to STK35—indicate that these kinases are broadly expressed in multiple tissues including liver, kidney, pancreas, spleen, thymus, prostate, placenta, heart, and brain (guo2003molecularcloningand pages 3-6). This widespread tissue distribution suggests that STK35 may participate in additional nuclear events in somatic cells, such as the regulation of transcription, DNA repair, and cell cycle progression. The ability of STK35 to phosphorylate histones further implicates it in the control of chromatin condensation and structure, which in turn can influence the accessibility of DNA to transcription factors and other regulatory proteins.  
   Furthermore, the role of STK35 in modulating nuclear signaling pathways extends to potential interactions with other key modulators of gene expression. Even though the full spectrum of substrate proteins and interacting partners for STK35 has not been completely characterized, current data support a model in which STK35 functions as an integrator of nuclear signals, modulating processes that range from transcriptional regulation to the stress response. This integration is made possible by its strategic nuclear localization and by its involvement in phosphorylation events that serve as molecular switches regulating the binding affinities and activities of downstream effectors.
8. Other Comments  
   STK35 is of significant biological interest given its essential involvement in nuclear signaling and chromatin dynamics. The severe reproductive phenotypes observed upon its disruption underscore the kinase’s critical role in spermatogenesis, and by extension, suggest that alterations in STK35 activity could contribute to reproductive disorders. Although there are currently no selective small molecule inhibitors that target STK35 specifically, the identification of key regulatory phosphorylation sites—such as those at S941 and S37—and its documented interactions with molecular chaperones provide promising avenues for the future development of targeted therapeutic agents (schwein2020theoglcnacmodification pages 27-28).  
   In addition, the discovery that its paralog PDIK1L exhibits a broad tissue expression pattern indicates that STK35 may exert functions beyond male reproductive biology. Its potential roles in cell cycle control, transcriptional regulation, and DNA repair render STK35 an intriguing subject for further investigations using advanced phospho-proteomic methods and high-resolution structural analyses. Currently, research in this area is focused on systematically mapping the spectrum of STK35 substrates and elucidating its network of interacting partners. Understanding these relationships is expected to provide deeper insights into the kinase’s regulatory mechanisms and its contributions to cellular homeostasis in both normal and disease states. No specific mutations or disease-associated variants have been prominently reported to date; however, ongoing studies aimed at identifying potential genetic alterations in patient cohorts may reveal additional clinical implications associated with dysregulated STK35 function.
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