1. Phylogeny  
   LIM domain kinase 2 (LIMK2) is a member of the LIM kinase family that is evolutionarily conserved across vertebrates, including mammals (human, mouse, rat), amphibians (e.g., Xenopus laevis), birds, and even certain invertebrates such as Drosophila species and Anopheles, although its distribution is largely confined to organisms requiring sophisticated cytoskeletal regulation. LIMK2 shares approximately 50% overall sequence identity with its paralog LIMK1 and is most closely related to TESK kinases, which also regulate cofilin activity (goyal2005dualfunctionof pages 24-28, scott2010limkinaseregulationa pages 16-20). Phylogenetic analyses indicate that LIMK2 emerged through gene duplication events early in vertebrate evolution and forms part of the kinome “core” set of regulatory enzymes that have been maintained to control actin dynamics (ribba2022theroleof pages 1-2, scott2010limkinaseregulation pages 11-16).
2. Reaction Catalyzed  
   LIMK2 catalyzes the transfer of the γ-phosphate group from ATP to specific serine and, in some contexts, tyrosine residues in its substrates. The canonical reaction can be written as:  
   ATP + protein-(L-serine or L-threonine) → ADP + protein-(L-serine/threonine)-phosphate + H⁺.  
   A primary physiological target of LIMK2 is the actin depolymerizing factor cofilin, which is phosphorylated at serine 3; this modification inactivates cofilin’s actin-severing activity, resulting in stabilization of actin filaments (brion2021limkinasesin pages 2-4).
3. Cofactor Requirements  
   The catalytic activity of LIMK2 is dependent on ATP as the phosphate donor and requires divalent metal ions—most commonly Mg²⁺—to facilitate nucleotide binding and proper orientation of the ATP for phosphotransfer reactions. These cofactor requirements are typical of serine/threonine kinases and support LIMK2’s function in phosphorylating substrates such as cofilin (brion2021limkinasesin pages 2-4, goyal2005dualfunctionof pages 24-28).
4. Substrate Specificity  
   LIMK2 exhibits a pronounced substrate specificity for members of the ADF/cofilin family, phosphorylating them on a specific serine residue (serine 3). This phosphorylation event blocks the actin filament–severing activity of cofilin, thereby controlling actin filament turnover and stabilization. Although detailed consensus motifs are not as comprehensively defined as for some other kinases, LIMK2 displays a marked preference for sequences that present the cofilin N-terminus in a conformation amenable to phosphorylation (chatterjee2022structuralaspectsof pages 10-11, ribba2022theroleof pages 13-15).
5. Structure  
   LIMK2 contains a modular domain organization that is essential for its function and regulation. The protein architecture comprises two N-terminal LIM domains—each containing double zinc finger motifs that mediate protein–protein interactions and provide structural stabilization—followed by a central PDZ domain that contributes to subcellular localization and further protein binding. Adjacent to the PDZ domain is a serine/proline-rich (S/P) region that may serve as a flexible linker and regulatory module, leading into the C-terminal kinase domain responsible for catalytic activity (goyal2005dualfunctionof pages 24-28, villalonga2023limkinaseslimk1 pages 1-2).  
   Structural studies and homology modelling indicate that the kinase domain adopts the characteristic bilobal architecture seen in protein kinases, with a smaller N-terminal lobe featuring a glycine-rich P loop, a larger C-terminal lobe, and an activation loop that undergoes phosphorylation-dependent transitions between inactive (DFG-out) and active (DFG-in) conformations (chatterjee2022structuralaspectsof pages 8-10, mittelstaedt2012structuralandfunctional pages 38-46). A unique feature of LIMK2 is the presence of a distinctive catalytic loop motif (DLNSHN) in subdomain VIB, which is rarely encountered among typical serine/threonine kinases and may contribute to its substrate specificity and regulation (manetti2012limkinasesare pages 3-6, sooreshjani2021identifyingtheversatile pages 23-28). Additionally, LIMK2 has been reported to form homodimers through interactions involving its LIM domains, a process that facilitates autophosphorylation and stabilization of the active kinase conformation (chatterjee2022structuralaspectsof pages 11-12, goyal2005dualfunctionofa pages 24-28).
6. Regulation  
   LIMK2 activity is tightly regulated by multiple mechanisms that ensure spatial and temporal control of actin cytoskeleton remodeling. One critical mechanism is phosphorylation of the activation loop; most notably, phosphorylation at threonine 505 by upstream kinases such as ROCK (Rho-associated protein kinase), p21-activated kinases (PAKs), and MRCKα serves to enhance LIMK2 kinase activity (brion2021limkinasesin pages 2-4, chatterjee2022structuralaspectsof pages 11-12). In addition, Aurora-A kinase has been identified as an activator of LIMK2, capable of phosphorylating multiple serine and threonine residues, which further modulate its activity (rak2014novellimk2inhibitor pages 1-2).  
   Autoinhibitory interactions also play a role in regulating kinase activity; the N-terminal LIM domains and the adjacent PDZ domain form intramolecular contacts that can restrain kinase activity until appropriate upstream signals trigger a conformational change (goyal2005dualfunctionof pages 24-28, ribba2022theroleof pages 12-13). Furthermore, stabilization and proper dimerization—a process promoted by interaction with molecular chaperones such as Hsp90—are essential for full kinase activation; inhibition of Hsp90 leads to decreased LIMK2 levels and consequent reduction in cofilin phosphorylation (chatterjee2022structuralaspectsof pages 1-3, manetti2012limkinasesare pages 3-6). Additional layers of regulation are provided by phosphatases, including PP1 and PP2A, which can dephosphorylate both LIMK2 and its substrates to restore basal actin dynamics (brion2021limkinasesin pages 2-4).
7. Function  
   LIMK2 plays an essential role in regulating actin filament dynamics through its ability to phosphorylate and inactivate cofilin, thereby modulating the balance between actin polymerization and depolymerization. The phosphorylation of cofilin stabilizes filamentous actin, contributing to proper cell morphology, migration, and adhesion (brion2021limkinasesin pages 2-4, chatterjee2022structuralaspectsof pages 10-11). Beyond its canonical role in actin dynamics, LIMK2 has been implicated in the regulation of microtubule dynamics; for instance, its phosphorylation of TPPP affects tubulin polymerization and is involved in the orientation of the mitotic spindle during early mitosis, thereby influencing cell division and cytokinesis (brion2021limkinasesin pages 2-4, podkowa2010characterizationofbmp pages 65-70).  
   LIMK2 is also involved in processes beyond direct cytoskeletal remodeling. It functions downstream of Rho family GTPases, thus integrating extracellular signals that govern cell migration, proliferation, and differentiation. Its activity has been linked to oncogenic signaling pathways in various cancers, where dysregulated actin remodeling can promote tumor invasion and metastasis (rak2014novellimk2inhibitor pages 1-2, ribba2022theroleof pages 13-15). In addition, LIMK2 has a role in suppressing ciliogenesis by phosphorylating CFL1, by controlling the directional trafficking of ciliary vesicles, and by promoting the nuclear localization of YAP1, where it functions as a transcriptional corepressor for genes such as AURKA and PLK1 (Protein Information section). Expression of LIMK2 is ubiquitous, with a broader tissue distribution compared to LIMK1, which is more restricted; this suggests that LIMK2 may have functions in multiple cell types ranging from neuronal to muscle and epithelial cells (villalonga2023limkinaseslimk1 pages 1-2, shah2023limk2amultifaceted pages 1-3).
8. Other Comments  
   Several small-molecule inhibitors targeting LIMK2 have been developed in preclinical models, with compounds such as T56-LIMKi demonstrating selective inhibition of LIMK2 activity without impairing LIMK1 function (rak2014novellimk2inhibitor pages 1-2). These inhibitors have been used to reduce phosphorylated cofilin levels and consequently affect tumor cell migration and proliferation, highlighting LIMK2 as a promising target in cancer therapy. Disease associations for LIMK2 include its implication in various cancers, where its dysregulated activity contributes to tumor growth, metastasis, and chemoresistance. Furthermore, LIMK2 is involved in cell cycle regulation during mitosis through its roles in astral microtubule organization and spindle orientation; defects in these processes may be associated with developmental abnormalities and proliferative disorders (brion2021limkinasesin pages 2-4, shah2023limk2amultifaceted pages 13-16). Beyond cancer, LIMK2’s suppression of ciliogenesis via multiple pathways also connects it to disorders of ciliopathy. No specific disease-causing mutations have been consistently reported in the literature for LIMK2; however, alterations in its regulation or expression levels have been linked to pathological conditions (chatterjee2022structuralaspectsof pages 11-12, ribba2022theroleof pages 13-15).
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