1. Phylogeny  
   Serine/threonine‐protein kinase PAK4 (p21‐activated kinase 4) is a member of the p21‐activated kinase (PAK) family, which is part of the larger STE20 kinase group. PAK4 is classified as a Group II PAK, in contrast to Group I members (PAK1–PAK3) that share distinct regulatory and structural features. Orthologs of PAK4 are found throughout metazoans, demonstrating its evolutionary conservation from lower eukaryotes to humans, and it is frequently considered within an evolutionary core set of kinases involved in cytoskeletal and survival signaling (li2022recentadvanceson pages 1-2, rane2014p21activatedkinases pages 1-2, dummler2009pakproteinkinases pages 17-21).
2. Reaction Catalyzed  
   PAK4 catalyzes the transfer of the γ‐phosphate group from ATP to specific serine and threonine residues on target substrate proteins. This enzymatic reaction is summarized as follows: ATP + [protein]–(L‐serine or L‐threonine) → ADP + [protein]–(L‐serine/threonine)‐phosphate + H⁺ (li2022recentadvanceson pages 1-2, crawford2012p21activatedkinaseinhibitors pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of PAK4, as is typical for serine/threonine kinases, depends on divalent metal ions, with Mg²⁺ serving as a necessary cofactor for ATP coordination and subsequent phosphoryl transfer (li2022recentadvanceson pages 1-2, dummler2009pakproteinkinases pages 17-21).
4. Substrate Specificity  
   PAK4 displays substrate specificity for serine/threonine residues embedded within motifs found on proteins that regulate cytoskeletal organization and cell adhesion. Functionally, PAK4 phosphorylates and inactivates the phosphatase SSH1, which results in increased inhibitory phosphorylation of cofilin, a key regulator of actin filament depolymerization (li2022recentadvanceson pages 1-2, dan2001cytoskeletalchangesregulated pages 1-2). Additionally, PAK4 phosphorylates LIM kinase 1 (LIMK1); phosphorylation of LIMK1 further contributes to cofilin inhibition and stabilization of actin structures (li2022recentadvanceson pages 7-10). In processes that regulate cell motility, PAK4 phosphorylates integrin beta5/ITGB5 and ARHGEF2, with the latter leading to activation of RHOA and subsequent regulation of focal adhesions and actin stress fiber assembly (li2022recentadvanceson pages 7-10, li2022recentadvanceson pages 16-16). Although a definitive consensus motif for PAK4 has not been fully established, biochemical profiling studies indicate a preference for serine phosphorylation in a context that may include nearby basic amino acids and hydrophobic residues, as observed in substrate analyses using peptide arrays (rennefahrt2007specificityprofilingof pages 3-4, miller2019comprehensiveprofilingof pages 7-9).
5. Structure  
   PAK4 exhibits a modular domain organization that underlies its regulatory and catalytic functions. Its N-terminal region comprises a p21-binding domain (PBD) that mediates interaction with small Rho GTPases such as CDC42 and, to a lesser extent, RAC1 (li2022recentadvanceson pages 1-2, rane2014p21activatedkinases pages 2-5). Adjacent to the PBD is an autoinhibitory or pseudosubstrate (PS/AID) domain, which keeps the kinase domain in an inactive conformation by obstructing substrate access until an activating signal is received (li2022recentadvanceson pages 1-2, rane2014p21activatedkinases pages 2-5). A basic residue cluster follows, contributing to membrane targeting. The C-terminal segment is occupied by the catalytic kinase domain, which displays the canonical bilobed structure seen in serine/threonine kinases. This domain contains key structural features such as the glycine-rich loop (responsible for ATP binding), the activation loop with the critical phosphorylation site (notably Ser474, which is constitutively phosphorylated yet held in an inhibited conformation due to the PS domain), the catalytic loop (featuring the HRD motif), and the DFG motif that coordinates the divalent ion cofactor (li2022recentadvanceson pages 1-2, li2022recentadvanceson pages 7-10, crawford2012p21activatedkinaseinhibitors pages 1-2). Structural studies have revealed conformational plasticity within the kinase domain, including a flexible back pocket that has been exploited in the design of isoform‐selective inhibitors (li2022recentadvanceson pages 16-16, rane2014p21activatedkinases pages 2-5).
6. Regulation  
   The activity of PAK4 is tightly regulated through multiple mechanisms. Autoinhibition is a key regulatory mechanism wherein the autoinhibitory pseudosubstrate domain binds intramolecularly to the kinase catalytic domain, thereby preventing substrate access despite the constitutive phosphorylation at Ser474 (li2022recentadvanceson pages 1-2, rane2014p21activatedkinases pages 2-5). Binding of GTP-bound CDC42 (and to a lesser extent RAC1) to the p21-binding domain induces a conformational change that alleviates autoinhibition, resulting in full activation of PAK4 (li2022recentadvanceson pages 1-2, rane2014p21activatedkinases pages 2-5, won2019pak4signalingin pages 2-3). Additional regulatory control can be mediated by interactions with SH3 domain–containing proteins—for instance, Src—and by changes in subcellular localization, which further modulate kinase activity (rane2014p21activatedkinases pages 2-5, won2019pak4signalingin pages 2-3). Although phosphorylation at the activation loop is constitutive, conformational regulation remains the dominant control mechanism, ensuring that PAK4 activity is only unleashed in response to appropriate upstream signals (li2022recentadvanceson pages 1-2, miller2019comprehensiveprofilingof pages 16-18).
7. Function  
   PAK4 plays multifaceted roles in cellular signaling, primarily through its effects on the cytoskeleton, cell adhesion, motility, and survival. One of its central functions is the modulation of actin filament dynamics. By phosphorylating and inactivating the protein phosphatase SSH1, PAK4 indirectly leads to increased inhibitory phosphorylation of cofilin; since cofilin is responsible for actin depolymerization, its inhibition results in the stabilization of actin filaments (li2022recentadvanceson pages 1-2, dan2001cytoskeletalchangesregulated pages 1-2). Moreover, PAK4 directly phosphorylates LIMK1, a kinase that further phosphorylates cofilin, thereby reinforcing the inhibition of actin depolymerization and contributing to the reorganization of the actin cytoskeleton (li2022recentadvanceson pages 7-10).  
   In addition to its role in modulating actin dynamics, PAK4 phosphorylates integrin beta5 (ITGB5); this modification plays a part in regulating cell motility and adhesion by influencing the formation and turnover of focal adhesions (li2022recentadvanceson pages 7-10, li2022recentadvanceson pages 16-16). PAK4 also targets ARHGEF2, leading to the activation of RHOA, which is critical for the assembly of focal adhesions and the formation of actin stress fibers (li2022recentadvanceson pages 7-10, li2022recentadvanceson pages 16-16). Furthermore, PAK4 contributes to cell survival pathways. It phosphorylates effectors linked to the apoptotic machinery, such as the BCL2 antagonist of cell death, thereby promoting cell survival and potentially contributing to oncogenic transformation (li2022recentadvanceson pages 1-2, li2022recentadvanceson pages 16-16, won2019pak4signalingin pages 2-3). PAK4 is frequently overexpressed or genomically amplified in various cancers—including pancreatic, breast, ovarian, and gastric carcinomas—underscoring its role in tumor cell proliferation, migration, invasion, and chemoresistance (li2022recentadvanceson pages 1-2, li2022recentadvanceson pages 16-16, won2019pak4signalingin pages 2-3, yu2022thesignificanceof pages 10-12). Its expression is not restricted to malignant tissues; PAK4 is ubiquitously expressed and is essential for normal embryonic and neural development, as evidenced by the embryonic lethality observed in PAK4‐null mice (rane2014p21activatedkinases pages 1-2, dummler2009pakproteinkinases pages 17-21).
8. Other Comments  
   Several small molecule inhibitors targeting PAK4 have been developed, with a particular emphasis on achieving isoform selectivity because pan‐PAK inhibition can lead to adverse effects such as cardiovascular toxicity. Notably, the allosteric inhibitor KPT-9274, which also targets NAMPT, has advanced to phase I clinical trials and shows promise in attenuating oncogenic processes associated with PAK4 overactivation (li2022recentadvanceson pages 1-2, crawford2012p21activatedkinaseinhibitors pages 1-2). PAK4 gene amplification, located on chromosome 19q13.2, is frequently observed in aggressive cancer phenotypes and correlates with poor prognosis; however, specific disease mutations affecting PAK4 functionality have not been extensively characterized in the literature (li2022recentadvanceson pages 16-16, won2019pak4signalingin pages 1-2, yu2022thesignificanceof pages 2-4). Ongoing research employing peptide array profiling and mutagenesis is deepening the understanding of PAK4’s substrate specificity and may inform the design of more selective therapeutic inhibitors in the future (rennefahrt2007specificityprofilingof pages 3-4, miller2019comprehensiveprofilingof pages 16-18). Additionally, PAK4 has been implicated in immune evasion processes in tumors, further underscoring its potential as a target for novel cancer therapeutics (li2022recentadvanceson pages 14-15).
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