1. Phylogeny  
   Activated CDC42 kinase 1 (ACK1), also known as TNK2, is a member of the non‐receptor tyrosine kinase (NRTK) family that belongs to the ACK subfamily. It is evolutionarily conserved among metazoans, with orthologs reported in mammals, bovine species, Drosophila, and Caenorhabditis elegans. Comparative sequence analyses demonstrate that ACK1 shares a high degree of conservation in its catalytic and regulatory domains with its sister kinase TNK1, and it clusters with other kinases that evolved to integrate extracellular stimuli with intracellular signaling cascades (kan2023domainarchitectureof pages 1-3, prietoechague2011regulationofackfamily pages 1-2, mahajan2010shepherdingaktand pages 1-2). In the context of the human kinome, ACK1 is classified as a non‐receptor tyrosine kinase; its atypical domain architecture, including an N‐terminal sterile alpha motif (SAM) and a C‐terminal ubiquitin association (UBA) domain, distinguishes it from most other tyrosine kinases whose regulatory modules are generally less complex (kan2023domainarchitectureof pages 24-26, prietoechague2011regulationofackfamily pages 1-2).
2. Reaction Catalyzed  
   ACK1 catalyzes the ATP‐dependent transfer of a phosphate group to tyrosine residues on substrate proteins. This reaction may be formally described as:  
     ATP + protein (with target tyrosine) → ADP + protein‐phosphotyrosine + H⁺  
   In performing this reaction, ACK1 phosphorylates multiple substrates including key signaling mediators such as AKT1, which is phosphorylated on Tyr176; the androgen receptor (AR) on Tyr267 and Tyr363; WWOX on Tyr287; as well as substrates involved in actin cytoskeleton dynamics and receptor trafficking, such as WASL and MCF2 (mahajan2010shepherdingaktand pages 2-3, mahajan2013ack1tyrosinekinase pages 1-2).
3. Cofactor Requirements  
   Consistent with the catalytic mechanisms of tyrosine kinases, ACK1 requires ATP as the phosphate donor and exhibits cofactor dependency on divalent metal ions, primarily Mg²⁺. The presence of Mg²⁺ is critical for coordinating ATP in the active site of the kinase domain and enabling efficient phosphoryl transfer (gajiwala2013ack1activationand pages 11-11).
4. Substrate Specificity  
   ACK1 is specific for phosphorylating tyrosine residues, and its substrate specificity is exemplified by the phosphorylation of several key signaling proteins. Notable substrates include:  
     • AKT1, with phosphorylation at Tyr176 that is crucial for its activation independent of PI3K signaling;  
     • The androgen receptor (AR), phosphorylated at Tyr267 and Tyr363, which facilitates its recruitment to androgen‐responsive enhancers; and  
     • WWOX, where phosphorylation at Tyr287 leads to regulation of its tumor suppressor function.  
   Additional substrates include proteins that participate in cytoskeletal reorganization and endocytic trafficking (e.g., WASL and MCF2). The substrate recognition appears to be influenced not only by the catalytic domain but also by interactions mediated by the SH3 domain, which can recognize proline‐rich motifs in target proteins. These substrate preferences ensure that ACK1 is integrated into diverse signaling circuits controlling cell migration, survival, and receptor dynamics (mahajan2010shepherdingaktand pages 2-3, kan2023domainarchitectureof pages 18-20).
5. Structure  
   ACK1 exhibits a modular domain organization that underpins both its catalytic activity and its regulation. At the N‐terminus, ACK1 contains a sterile alpha motif (SAM) domain that is predicted to fold into a structure composed of four short α-helices and a longer C-terminal helix. This domain is implicated in homodimerization and membrane targeting, processes that are essential for full kinase activation (kan2023domainarchitectureof pages 1-3).

The central region of ACK1 is dominated by a conserved tyrosine kinase domain (KD) that adopts the typical bilobed structure seen in many protein kinases. The N‐lobe is primarily composed of β‐strands and contains the glycine-rich P-loop, whereas the C‐lobe is mainly α‐helical and houses the activation loop, which, in ACK1, contains the critical autophosphorylation site Tyr284. Structural studies reveal that the kinase domain of ACK1 also features a well‐defined DFG motif, an invariant catalytic lysine, and hydrophobic spines (both catalytic [C‐spine] and regulatory [R‐spine]) that are essential for stabilizing the active conformation (kan2023domainarchitectureof pages 14-16, kan2023domainarchitectureof pages 28-29).

Downstream of the kinase domain, ACK1 contains a Src Homology 3 (SH3) domain. Unusually, this SH3 domain is located C‐terminally with respect to the kinase domain as opposed to the more common N‐terminal positioning in other tyrosine kinases. Structurally, the SH3 domain of ACK1 forms a compact β-barrel structure and is involved in binding proline-rich sequences, potentially mediating protein–protein interactions that regulate substrate recognition and allosteric control (kan2023domainarchitectureof pages 29-30, mahajan2010shepherdingaktand pages 3-4).

In addition, ACK1 harbors a Cdc42/Rac interactive binding (CRIB) domain; this domain is relatively short, yet it is unique in that it is found in a tyrosine kinase and enables direct binding to the GTP-bound form of CDC42. This interaction is important for linking ACK1 activity to small GTPase signaling cascades and facilitating cytoskeletal regulation. Further toward the C-terminus lies a Mig6 homology region (MHR), which is thought to play an autoinhibitory role by interacting with the kinase and SH3 domains, thereby modulating ACK1 activation. Finally, at the extreme C‐terminus, ACK1 contains a ubiquitin association (UBA) domain. This UBA domain is unusual among kinases in that it directly binds polyubiquitin chains, a feature that has implications for the stability and turnover of ACK1 as well as for its role in receptor trafficking (kan2023domainarchitectureof pages 24-26, kan2023domainarchitectureof pages 32-33, mahajan2013ack1tyrosinekinase pages 1-2).

1. Regulation  
   The activity of ACK1 is tightly regulated through a combination of autoinhibitory interactions, phosphorylation events, and protein–protein interactions. One central mechanism involves the Mig6 homology region (MHR), which interacts intramolecularly with the kinase and SH3 domains to maintain ACK1 in an autoinhibited conformation under basal conditions. Activation is achieved when extracellular signals, such as those delivered via receptor tyrosine kinases (e.g., EGFR, PDGFR, HER2), initiate conformational changes that disrupt these inhibitory interactions. This relief of autoinhibition is accompanied by autophosphorylation at key residues, most notably Tyr284 in the activation loop, which is critical for full catalytic activity (mahajan2010shepherdingaktand pages 2-3, mahajan2013ack1tyrosinekinase pages 2-4).

Additional layers of regulation are provided by interactions with small GTPases such as CDC42, which bind the CRIB domain and further modulate the kinase’s conformational state as well as its substrate specificity. Dimerization mediated by the SAM domain has also been shown to be important for autoactivation, thereby linking membrane localization and oligomerization to functional output (kan2023domainarchitectureof pages 1-3, kan2023domainarchitectureof pages 24-26).

ACK1 is subject to regulation by ubiquitination. Its UBA domain facilitates binding to polyubiquitin, and interactions with E3 ubiquitin ligases such as Nedd4-1 and Nedd4-2 target ACK1 for ubiquitin-mediated degradation. This post-translational modification serves to control protein stability and terminate signaling once the appropriate cellular response has been achieved (prietoechague2011regulationofackfamily pages 4-5, mahajan2010shepherdingaktand pages 2-3). In some cellular contexts, binding to 14-3-3 proteins via phosphorylated motifs may further sequester ACK1 and modulate its subcellular localization, although the precise details of this regulation require further elucidation (balasooriyage2024integratingclinicalcancer pages 2-2). Thus, ACK1 regulation is a multifaceted process that integrates signaling cues through phosphorylation, dimerization, and ubiquitin-mediated turnover to finely tune its kinase activity and downstream signaling outcomes.

1. Function  
   ACK1 plays a central role in transducing extracellular signals to both cytosolic and nuclear effectors, thereby influencing a broad spectrum of cellular processes. Its kinase activity is essential for the regulation of cell spreading, migration, survival, growth, and proliferation. Through phosphorylation of its substrates, ACK1 affects multiple signaling pathways that govern these processes. Key substrates include:

  • AKT1: Phosphorylation of AKT1 at Tyr176 by ACK1 facilitates an alternative mode of AKT activation independent of PI3K, thereby promoting cell survival and proliferation (mahajan2010shepherdingaktand pages 2-3, liu2019researchprogressof pages 5-6).  
  • Androgen Receptor (AR): ACK1 phosphorylates the androgen receptor on Tyr267 and Tyr363. This modification promotes the recruitment of AR to androgen-responsive enhancers (AREs), thus influencing transcriptional programs associated with androgen-independent prostate cancer progression (mahajan2010shepherdingaktand pages 2-3, mahajan2015ack1tnk2tyrosinekinase pages 1-2).  
  • WWOX: Phosphorylation of the tumor suppressor WWOX on Tyr287 by ACK1 alters its stability through enhanced polyubiquitination and degradation, thereby modulating its anti-proliferative effects (mahajan2010shepherdingaktand pages 2-3, mahajan2013ack1tyrosinekinase pages 15-17).  
  • WASL (Wiskott–Aldrich Syndrome-like protein): By phosphorylating components involved in actin cytoskeletal dynamics, ACK1 participates in the reorganization of the cytoskeleton that is critical for cell migration (kan2023domainarchitectureof pages 18-20).  
  • BCAR1: Acting as a downstream effector of CDC42, ACK1 phosphorylates BCAR1 to mediate CDC42-dependent cell migration (thaker2017activatedcdc42associatedkinase pages 20-25).

In addition, ACK1 is implicated in receptor trafficking and clathrin-mediated endocytosis. Through direct interactions with the epidermal growth factor receptor (EGFR) and clathrin, ACK1 regulates ligand-induced degradation of EGFR, leading to its accumulation at the early endosomal membrane. This function not only modulates EGFR signaling but also impacts the duration and intensity of downstream mitogenic signals (kan2023domainarchitectureof pages 26-28, wu2016thenonreceptortyrosine pages 1-2).

Tissue expression studies reveal that ACK1 is ubiquitously expressed, with particularly high levels observed in immune tissues (such as spleen and thymus), brain, and endocrine organs. Moreover, its role in modulating both cytosolic and nuclear signaling pathways implicates ACK1 in an array of physiological contexts ranging from cell motility and adhesion to neuronal development and synaptic plasticity (kan2023domainarchitectureof pages 7-9, thaker2017activatedcdc42associatedkinase pages 3-4). Collectively, ACK1 functions as an integrator of external signals by phosphorylating a diverse set of substrates, which in turn modulate processes central to cellular proliferation, migration, receptor turnover, and survival.

1. Other Comments  
   ACK1 has emerged as an attractive therapeutic target due to its prominent role in oncogenic signaling pathways. Aberrant activation of ACK1, which may result from gene amplification, somatic mutations (e.g., E346K in the kinase domain, D163E, or mutations in the UBA domain such as S985N), and dysregulation of its ubiquitin-mediated turnover, has been implicated in the progression of various cancers including prostate, breast, lung, colorectal, and ovarian cancers (mahajan2013ack1tyrosinekinase pages 15-17, prietoechague2011regulationofackfamily pages 7-8). Several small molecule inhibitors have been identified through structure-based drug design and high-throughput screening approaches. Compounds such as AIM-100 and dasatinib have been shown to inhibit ACK1 activity in preclinical models, with further lead compounds emerging from computational studies targeting critical active site residues (kumar2021identificationofack1 pages 8-9, lawrence2015developmentofnovel pages 1-3).

In addition to its role in cancer, ACK1 is implicated in the regulation of receptor trafficking and may contribute to alterations in endocytosis that underlie various disease states. Its interaction with polyubiquitin via the UBA domain and subsequent regulation by E3 ubiquitin ligases underscores a mechanism by which ACK1 activity and stability are coordinated, providing another point of therapeutic intervention (prietoechague2011regulationofackfamily pages 5-6, mahajan2010shepherdingaktand pages 2-3).

Recent studies in immunology have also elucidated a role for ACK1 in T-cell receptor signaling. For example, ACK1 has been shown to interact via its SAM domain with the adaptor protein SLP-76, resulting in phosphorylation of proximal tyrosine residues that are important for T-cell activation and IL-2 production. This suggests that apart from its oncogenic roles, ACK1 may also be a critical regulator of immune cell function, thereby expanding its potential impact on immunomodulatory therapies (thaker2017activatedcdc42associatedkinase pages 3-4).

Furthermore, ACK1’s ability to phosphorylate several substrates involved in hormone signaling (such as the androgen receptor) links it directly to cancers that are driven by steroid hormones, including castration-resistant prostate cancer. The dual roles in both receptor trafficking and nuclear receptor activation make ACK1 a unique signaling node with multifaceted potential for targeted drug development (mahajan2010shepherdingaktand pages 2-3, mahajan2015ack1tnk2tyrosinekinase pages 1-2).

Overall, the diverse regulatory mechanisms, substrate specificities, and biological functions of ACK1 underscore its importance as a pivotal enzyme in both normal physiology and disease. Its unique domain architecture, featuring an N-terminal SAM domain, a centrally located kinase domain with allosteric regulatory elements such as the hydrophobic spines and activation loop, together with a C-terminal SH3 and UBA domain, provide multiple surfaces for both intramolecular regulation and potential pharmacological targeting (kan2023domainarchitectureof pages 16-18, kan2023domainarchitectureof pages 26-28). These properties have driven significant interest in the development of specific inhibitors that may one day serve as therapeutics for a range of malignancies and other disorders associated with dysregulated ACK1 activity (lawrence2015developmentofnovel pages 1-3, kumar2021identificationofack1 pages 8-9, chouhan2023tnk2ack1mediatedphosphorylationof pages 1-2).

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