1. Phylogeny  
   Cyclin‐dependent kinase 14 (CDK14), also known as KIAA0834 or PFTK1, is a member of the cyclin‐dependent kinase family that falls within the CMGC group of the human kinome. CDK14 is evolutionarily conserved among metazoans, and orthologs have been detected from invertebrate species such as Drosophila to mammals, underscoring that this kinase subfamily emerged in an early ancestor of metazoans and has retained its role in the control of cell division over evolutionary time (bradley2019evolutionofprotein pages 1-2, briedis2008thedistributionand pages 205-208). Classic studies on the kinase complement of the human genome have established that CDK14, together with other members of the PFTAIRE subfamily, diverged early from the canonical cyclin‐dependent kinases; these kinases maintain conserved catalytic domains and cyclin-binding regions that are essential for their function in cell cycle regulation (krupa2002therepertoireof pages 2-3, bradley2019evolutionofprotein pages 19-21). In addition, subsequent analyses have demonstrated that CDK14 clusters with related PFTAIRE kinases, sharing significant sequence conservation within the kinase core that is indicative of their common origin and functional similarity in mediating phosphorylation events necessary for cell proliferation and developmental signaling (Manning et al. 2002, Manning et al. 2002, bradley2019evolutionofprotein pages 21-22). Thus, CDK14 is assigned to the CMGC group and more specifically to the cyclin‐dependent kinases that regulate cell cycle progression as well as transcription, and its tight conservation suggests a crucial role in eukaryotic biology (briedis2008thedistributionand pages 205-208).
2. Reaction Catalyzed  
   CDK14 catalyzes the ATP‐dependent phosphorylation reaction that transfers the gamma‐phosphate group from ATP to serine or threonine residues on substrate proteins. The chemical reaction can be summarized as:  
     ATP + [protein]-(L-serine or L-threonine) = ADP + [protein]-(L-serine/threonine)-phosphate + H⁺  
   This phosphotransfer reaction represents the fundamental enzymatic activity that defines serine/threonine kinases and enables CDK14 to modulate downstream signaling pathways through the reversible phosphorylation of target proteins (bradley2019evolutionofprotein pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of CDK14 is dependent on divalent metal ions, predominantly Mg²⁺, which are required to coordinate the binding of ATP in the active site of the enzyme. The presence of Mg²⁺ not only stabilizes the nucleotide but also facilitates the precise positioning of the gamma‐phosphate group for transfer to the substrate’s serine or threonine residue. This requirement for Mg²⁺ is a common characteristic of serine/threonine protein kinases and is critical for effective phosphoryl transfer during catalysis (bradley2019evolutionofprotein pages 21-22, kostich2002humanmembersof pages 11-12).
4. Substrate Specificity  
   The substrate specificity of CDK14 is defined by its ability to recognize serine/threonine phosphorylation sites within specific amino acid motifs. Data from the comprehensive atlas of substrate specificities for the human serine/threonine kinome indicate that CDK family kinases typically prefer proline‐directed motifs, meaning that a phosphorylatable serine or threonine residue is frequently positioned immediately upstream of a proline residue. In the context of CDK14, one experimentally validated target is the Wnt pathway co‐receptor LRP6, which is phosphorylated by CDK14 at Ser-1490—a modification that is consistent with a [S/T]P motif. Although an exact consensus sequence solely for CDK14 has not been fully delineated, its assignment to the cyclin‐dependent kinase family strongly suggests that the recognition pattern incorporates not only the proline adjacent to the phosphoacceptor but also additional flanking residues that may include basic amino acids or other determinants that help direct substrate binding (johnson2023anatlasof pages 10-11, alonso2021caracterizacióndecdk1418 pages 114-118, alrawi2023cyclinaand pages 13-15). Moreover, additional studies indicate that substrate specificity in related kinases is further refined by the interaction with activating cyclins that contribute to conformational changes in the active site, thus enhancing the selectivity for substrates that display the characteristic proline-directed amino acid pattern (johnson2023anatlasof pages 6-7, bradley2019evolutionofprotein pages 21-22).
5. Structure  
   CDK14 exhibits a canonical protein kinase structure that is typical of cyclin‐dependent kinases. The enzyme is characterized by a central kinase domain of approximately 250–300 amino acids that is partitioned into an N-terminal lobe and a larger C-terminal lobe. The N-terminal lobe is mainly composed of β-sheets and houses the glycine-rich loop, which plays a critical role in ATP binding. In contrast, the C-terminal lobe is predominantly α-helical and contains key catalytic elements including the catalytic loop, the activation loop (often referred to as the T-loop), and the C-helix. The activation loop is particularly important as it undergoes conformational changes upon phosphorylation and cyclin binding, thereby acting as a molecular switch that modulates access to the active site (faezov2023alphafold2modelsof pages 1-4, johnson2023anatlasof pages 4-5).

A defining structural feature of CDK14 is its hydrophobic spine, a series of conserved hydrophobic residues that stabilize the active conformation of the enzyme. In addition, the C-helix within the C-terminal lobe is oriented such that, upon proper cyclin interaction, it forms a critical salt bridge with a conserved lysine residue in the ATP-binding pocket. This alignment of the catalytic residues is essential for efficient phosphoryl transfer during the catalytic cycle (scheeff2005structuralevolutionof pages 10-11, kostich2002humanmembersof pages 4-5).

Furthermore, CDK14 contains a conserved cyclin-binding domain, which is responsible for its interaction with regulatory cyclins such as cyclin D3 (CCDN3) and, in some studies, cyclin Y. The binding of these cyclins induces conformational rearrangements that are necessary for the full activation of the kinase. Structural predictions from recent AlphaFold2 models have reinforced the notion that CDK14 adopts a typical CDK fold, complete with the expected catalytic cleft, activation segment, and regulatory regions, even though a dedicated high-resolution crystal structure for CDK14 has not yet been published (faezov2023alphafold2modelsof pages 20-23, faezov2023alphafold2modelsof pages 23-25).

1. Regulation  
   The regulatory mechanisms governing CDK14 activity are intrinsically linked to its association with cyclins and post-translational modifications. The binding of cyclin D3 (CCDN3) is essential for CDK14 activation, as cyclin association induces conformational changes that properly orient the activation loop and catalytic residues, thereby facilitating efficient substrate phosphorylation. In several studies, cyclin Y has also been implicated as an activating partner, providing further evidence of the versatility in cyclin-mediated regulation of CDK14 (alonso2021caracterizacióndecdk1418 pages 114-118, alrawi2023cyclinaand pages 32-35).

Post-translational modifications, particularly phosphorylation, play a pivotal role in modulating the activity of CDK14. Although specific phosphorylation sites within the activation loop of CDK14 are still under investigation, the regulatory paradigm mirrors that of other CDKs wherein phosphorylation acts as a switch that transitions the kinase from an inactive to an active state. In addition to phosphorylation, there is the potential for regulation through protein–protein interactions and ubiquitination events that may impact protein stability and turnover during distinct phases of the cell cycle (alrawi2023cyclinaand pages 17-20, reinhardt2023acriticalevaluation pages 22-23).

Furthermore, interactions with 14-3-3 proteins have been reported to influence the binding of cyclins, particularly with cyclin Y; such interactions are thought to enhance complex formation and may also affect the subcellular localization of the CDK14-cyclin complex, thereby fine-tuning the kinase’s activity in response to cellular signals (mikolcevic2012orphankinasesturn pages 2-3, alrawi2023cyclinaand pages 6-8). Collectively, these regulatory mechanisms ensure that CDK14 activity is precisely modulated in time and space during the progression of the cell cycle.

1. Function  
   CDK14 functions primarily as a serine/threonine–protein kinase that plays a significant role in the control of the eukaryotic cell cycle. One of its well-documented functions includes the phosphorylation of the Wnt co-receptor LRP6 at Ser-1490 during the G2/M phase, an event that is critical for the activation of canonical Wnt/β-catenin signaling. This phosphorylation event contributes to the regulation of cell proliferation and developmental processes (alonso2021caracterizacióndecdk1418 pages 114-118, alrawi2023cyclinaand pages 32-35).

In addition to modulating Wnt signaling, CDK14 interacts with cyclin D3 (CCDN3) to regulate cell cycle progression. In vitro studies have demonstrated that CDK14 is capable of phosphorylating the retinoblastoma protein (RB1), although the physiological relevance of this phosphoregulation in vivo remains to be conclusively established. Beyond its roles in cell cycle control, emerging evidence suggests that CDK14 may also participate in other cellular processes such as meiosis and neuronal differentiation. Furthermore, there is data indicating that CDK14 can indirectly modulate insulin-responsive glucose transport, adding a possible metabolic dimension to its functional repertoire (briedis2008thedistributionand pages 205-208, kostich2002humanmembersof pages 11-12).

Expression studies have shown that CDK14 is present in a variety of proliferative tissues, including those of the nervous system, gastrointestinal tract, and reproductive organs. The ubiquitous as well as tissue-specific expression pattern of CDK14 supports its critical role in coordinating cell division and signal transduction processes. By integrating signals from both cyclin partners and upstream regulatory pathways, CDK14 acts as a nodal point that influences not only cell cycle transitions but also the broader network of pathways that govern cell proliferation and differentiation (alonso2021caracterizacióndecdk1418 pages 114-118, karimbayli2024insightsintothe pages 1-2).

1. Other Comments  
   Although numerous inhibitors targeting members of the cyclin-dependent kinase family have been developed, there remains a lack of highly selective and clinically approved inhibitors specific for CDK14. Some covalent inhibitors designed against the broader TAIRE subfamily of CDKs exhibit activity against CDK14; however, these compounds typically show pan-TAIRE specificity and thus inhibit related kinases, complicating assessments of selectivity. As such, CDK14 currently represents an attractive candidate for further drug discovery research aimed at developing selective inhibitors (ferguson2019discoveryofcovalent pages 12-13, poll2024aresourcedatabase pages 6-9).

In terms of disease association, dysregulation of CDK14 has been implicated in aberrant cell cycle control and dysregulated Wnt signaling pathways—features that are commonly observed in various types of cancer. While specific disease mutations and their direct functional consequences remain to be extensively characterized, overexpression or hyperactivation of CDK14 has been observed in multiple tumor types, suggesting potential links to oncogenic processes. Continued research into the regulation and substrate interactions of CDK14 may eventually establish its utility as a therapeutic target in proliferative disorders and other pathological conditions (krupa2002therepertoireof pages 2-3, alrawi2023cyclinaand pages 32-35).

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