1. Phylogeny  
   Cyclin‐dependent kinase 16 (CDK16), also known as PCTAIRE1 or PCTK1, is a member of the PCTAIRE subfamily of cyclin‐dependent kinases, a group that is evolutionarily conserved across eumetazoans. Within the human kinome, CDK16 is grouped with related kinases CDK17 and CDK18, and it shows significant sequence similarity in the kinase domain to other CDKs such as CDK2 and CDK5. Based on the analyses by Manning et al. (2002) describing the protein kinase complement of the human genome and the evolutionary trajectory from yeast to mammals, CDK16 belongs to the CMGC group of serine/threonine kinases. Its orthologs are present in all mammalian species and more broadly in metazoans, while its unique PCTAIRE motif in the αC-helix distinguishes it from classical cell cycle regulators. This phylogenetic context underscores its placement in an evolutionary “core” set of kinases that emerged early in eukaryotic evolution, a set that includes not only members of the cell cycle machinery but also kinases with specialized functions in post-mitotic tissues such as the brain and testis (Manning2002Science, Manning2002Trends; karimbayli2024insightsintothe pages 1-2, mikolcevic2012orphankinasesturn pages 1-2).
2. Reaction Catalyzed  
   CDK16 catalyzes the transfer of a phosphate group from ATP to hydroxyl groups on serine or threonine residues in proteins. The canonical reaction can be represented as:  
     ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺  
   This reaction is typical of serine/threonine kinases in the cyclin-dependent kinase family and aligns with the reaction mechanisms described for related kinases (dixon2015structureandinhibition pages 23-28).
3. Cofactor Requirements  
   The catalytic activity of CDK16 is dependent upon the presence of divalent metal ions such as Mg²⁺, which are essential for coordinating ATP within the active site and enabling efficient phosphate transfer. In biochemical assays, Mg²⁺ (and in some instances Mn²⁺ as an alternative cofactor) is required to facilitate the kinase activity of CDK16, consistent with the cofactor dependencies reported for many CDK family members (dixon2015structureandinhibition pages 43-45, endicott2013structuralcharacterizationof pages 1-2).
4. Substrate Specificity  
   CDK16 is a serine/threonine kinase with substrate specificity generally aligned with the proline-directed phosphorylation pattern typical of CDKs. In particular, CDK16 can phosphorylate substrates that carry specific sequence motifs, although the exact consensus motif for CDK16 has not been fully elucidated. Experimentally, CDK16 phosphorylates substrates such as the N-ethylmaleimide sensitive factor (NSF), thereby regulating NSF oligomerization, and has been shown in vitro to phosphorylate cyclin Y at serine 336. In broader studies focusing on serine/threonine kinase substrate preferences, an atlas has been constructed that details the intrinsic specificity of many such kinases, where substrates typically exhibit a preference for a serine or threonine residue immediately followed by a proline. Additional insights also come from studies examining the intrinsic substrate specificity of tyrosine kinases, which, although not directly applied to CDK16, help establish the context of kinase substrate discrimination within the kinome; however, CDK16 remains defined as a serine/threonine kinase (Johnson2023Atlas pages 759-766, Yaron-Barir2024Atlas pages 1174-1181, xie2018cdk16phosphorylatesand pages 9-13).
5. Structure  
   CDK16 contains a centrally located kinase domain that is characteristic of the CDK family, flanked by distinctive N-terminal and C-terminal extensions. The kinase domain itself comprises the typical bilobal fold with an N-terminal lobe consisting mainly of β-sheets and a glycine-rich loop, and a larger C-terminal lobe that is predominantly α-helical. Key structural features include the DFG motif, which coordinates Mg²⁺ ions necessary for catalytic activity, and the PCTAIRE motif located within the αC-helix, which distinguishes CDK16 from classical CDKs that generally contain the PSTAIRE sequence. Crystal structures, such as those reported in studies using the inhibitor indirubin E804, reveal that in the absence of cyclin binding, CDK16 adopts an intrinsically inactive conformation with a displaced αC-helix and an activation loop in a “DFG-out” conformation; however, binding of the regulatory partner cyclin Y induces a conformational shift toward an active kinase state, as the cyclin interaction facilitates proper alignment of the catalytic residues (dixon2015structureandinhibition pages 147-151, endicott2013structuralcharacterizationof pages 3-5, karimbayli2024insightsintothe pages 2-4). In addition, the N-terminal extension harbors regulatory elements including a phosphorylation site at serine 153, which plays a key role in modulating the binding of cyclin Y. The overall three-dimensional organization of CDK16 thus features a conserved catalytic core with unique regulatory appendages that are critical for its isoform-specific interactions and subcellular targeting (mikolcevic2012orphankinasesturn pages 2-3, dixon2015structureandinhibition pages 28-33).
6. Regulation  
   Regulation of CDK16 occurs primarily through its association with cyclin Y, which is essential for kinase activation. In its monomeric form, CDK16 is catalytically inactive, and cyclin binding is required to induce the conformational rearrangements necessary for active substrate phosphorylation. A critical regulatory event is the phosphorylation of CDK16 at serine 153, a residue that is part of a consensus sequence for protein kinase A (PKA). Phosphorylation at this site negatively regulates the interaction between CDK16 and cyclin Y, thereby inhibiting kinase activity. In certain cellular contexts, a decrease in serine 153 phosphorylation permits the formation of an active CDK16–cyclin Y complex; this regulatory mechanism is particularly evident in testicular tissue, where lower levels of phosphorylated serine 153 correlate with active CDK16 required for spermatogenesis (mikolcevic2012orphankinasesturn pages 10-11, karimbayli2024insightsintothe pages 17-18). Additionally, allosteric regulation through cyclin binding induces rearrangement of the C-helix and the activation loop, facilitating ATP binding and substrate recognition. Other regulatory mechanisms, including interactions with proteins such as 14-3-3 and potential phosphorylation by additional kinases such as CDK5/p35, contribute further to the fine-tuning of CDK16 activity in neuronal cells and during vesicle trafficking events (hernandezortega2019phosphoregulationofthe pages 5-7, janackova2023mechanismusregulacecyklindependentní pages 20-24).
7. Function  
   CDK16 plays diverse roles in cellular physiology that encompass vesicle-mediated transport, exocytosis, and the regulation of hormone release in the brain. One of its critical functions is the phosphorylation of NSF, a key regulatory step in controlling NSF oligomerization. This activity is important for vesicular trafficking and exocytosis in neuronal cells, where CDK16 regulates the release of growth hormone (GH1) by brain neurons. Beyond its role in exocytosis, CDK16 is required for normal spermatogenesis; its activity is crucial in postmeiotic spermatids, and loss of CDK16 function in animal models results in impaired sperm differentiation and fertility defects. CDK16 is also implicated in neuronal differentiation and dendrite development, possibly by modulating components of the cytoskeletal and vesicle trafficking machinery, and it has been associated with the regulation of insulin secretion in pancreatic beta-cells in response to changes in blood glucose levels. In vitro, CDK16 has been shown to phosphorylate cyclin Y at serine 336, further highlighting its capacity to regulate components of its own activation complex (amrhein2022discoveryof3amino1hpyrazolebased pages 1-3, amrhein2022discoveryof3amino1hpyrazolebased pages 3-4, xie2018cdk16phosphorylatesand pages 13-13, li2022cdk16promotesthe pages 18-18, mikolcevic2012cyclindependentkinase16pctaire pages 2-3).
8. Other Comments  
   Several small-molecule inhibitors have been developed or identified that target CDK16 activity. For instance, selective 3-amino-1H-pyrazole-based compounds have been reported to inhibit CDK16 with high potency; compound 43d is one such inhibitor that has been used to illuminate the biological functions of the PCTAIRE kinase family (amrhein2022discoveryof3amino1hpyrazolebased pages 1-3). Additionally, broader-spectrum kinase inhibitors such as dabrafenib and rebastinib, originally developed for other kinase targets, have been shown to inhibit CDK16 activity in various cellular assays (dixon2015structureandinhibition pages 147-151). CDK16’s involvement in critical processes such as vesicle trafficking, neuronal differentiation, and spermatogenesis, as well as its potential role in the regulation of insulin secretion, makes it an attractive target for therapeutic intervention in disorders ranging from infertility to certain neurodegenerative conditions and metabolic syndromes. However, while the inhibition of CDK16 may offer clinical benefits, the specificity of available inhibitors is a matter of ongoing research, and further work is required to develop compounds that selectively target CDK16 without affecting other cyclin-dependent kinases (pepino2021overviewofpctk3cdk18 pages 17-18, dixon2015structureandinhibition pages 40-43).
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