1. Phylogeny  
   Cyclin-dependent kinase 18 (CDK18), also known as PCTAIRE3 or PCTK3, is a member of the CDK family that falls within the atypical PCTAIRE subfamily. Unlike classical CDKs that harbor the highly conserved PSTAIRE sequence for cyclin binding, CDK18 contains a variant “PCTAIRE” motif. This subgroup also includes CDK16 (PCTK1) and CDK17 (PCTK2), and phylogenetic studies indicate that CDK18 and the other PCTAIRE kinases are evolutionarily conserved from simple invertebrates such as Caenorhabditis elegans to mammals, although CDK18 itself appears later in evolution compared to some other family members (alonso2021caracterizacióndecdk1418 pages 29-32, pepino2021overviewofpctk3cdk18 pages 2-3). Phylogenetic analyses based on the human kinome reveal that CDK18 is most closely related to other kinases within the CDK5-related branch and is part of a core set that emerged early in eukaryotic evolution. Its evolutionary relationship with canonical CDKs is marked by conserved catalytic features, yet the divergence in its cyclin-binding motif underscores its distinct regulatory and functional evolution (pepino2021overviewofpctk3cdk18 pages 2-3, karimbayli2024insightsintothe pages 1-2).
2. Reaction Catalyzed  
   CDK18 functions as a serine/threonine kinase that catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of serine or threonine residues on substrate proteins. The biochemical reaction follows the general scheme: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺. This phosphorylation event is an essential regulatory step in signal transduction cascades, affecting protein function, localization, and interaction (malumbres2014cyclindependentkinases pages 3-5).
3. Cofactor Requirements  
   As with most protein kinases, the catalytic activity of CDK18 depends on the presence of divalent metal ions, with Mg²⁺ being the primary cofactor required for proper ATP coordination and phosphotransfer activity. The Mg²⁺ ion functions to stabilize the negative charges on the phosphate groups of ATP, thereby facilitating the nucleophilic attack required for the phosphorylation reaction (malumbres2014cyclindependentkinases pages 3-5).
4. Substrate Specificity  
   CDK18 phosphorylates serine/threonine residues on protein substrates. In vitro kinase assays have demonstrated that CDK18 is capable of phosphorylating the retinoblastoma protein (Rb) when activated by cyclin A2, though the phosphorylation efficiency is lower compared to canonical kinases such as CDK2 (matsuda2014pctairekinase3cyclindependent pages 1-2, matsuda2014pctairekinase3cyclindependent pages 10-11). Although a defined consensus substrate motif for CDK18 has not been conclusively established in the literature, experimental data suggest that the kinase, like other serine/threonine kinases, may recognize substrates in a sequence context that favors basic residues upstream of the phosphorylation site. In addition, the functional involvement of CDK18 in cellular processes such as replication stress response and actin cytoskeleton dynamics implies that its substrate repertoire might include proteins implicated in DNA damage checkpoint regulation (barone2016humancdk18promotes pages 1-1) as well as regulators of cytoskeletal organization (pepino2021overviewofpctk3cdk18 pages 9-11).
5. Structure  
   CDK18 exhibits a domain organization characteristic of cyclin-dependent kinases with a central catalytic domain flanked by regulatory extensions. The catalytic kinase domain, approximately 250–300 amino acids in length, is comprised of a bilobal structure with an N-terminal lobe predominantly featuring β-sheets and a glycine-rich loop, and a larger C-terminal lobe enriched in α-helices. Within this conserved domain, key motifs are present, including a modified cyclin-binding αC-helix that contains the PCTAIRE motif instead of the canonical PSTAIRE sequence, a feature that distinguishes CDK18 from cell cycle CDKs (alonso2021caracterizacióndecdk1418 pages 32-35, pepino2021overviewofpctk3cdk18 pages 2-3).  
   The activation loop, which generally extends from a conserved DFG motif to an APE motif, is presumed to participate in regulating access to the catalytic cleft and substrate binding, similarly to other CDKs. Although no high-resolution crystal structure has been reported specifically for CDK18, comparative modeling based on the structure of closely related PCTAIRE kinases (such as CDK16) suggests that CDK18 maintains the overall kinase fold. Structural studies of related kinases have revealed a DFG-in conformation correlating with the active state, and it is anticipated that CDK18 exhibits a similar conformation upon activation via cyclin binding and post-translational modifications (endicott2013structuralcharacterizationof pages 2-3, karimbayli2024insightsintothe pages 2-4).  
   In addition to its catalytic core, CDK18 contains unique N-terminal and C-terminal regions that are less conserved and implicated in mediating protein–protein interactions and subcellular localization. These extensions may contribute to the regulation of substrate specificity and the interaction with distinct cyclin partners, notably cyclin A2, which is essential for its kinase activity (pepino2021overviewofpctk3cdk18 pages 2-3, matsuda2014pctairekinase3cyclindependent pages 2-3). Furthermore, structural models indicate that CDK18 preserves characteristic catalytic residues, such as a conserved lysine in the ATP-binding pocket that is critical for catalytic activity, and an intact DFG motif for metal ion coordination (kamkar2015pftaire1(cyclindependent pages 29-34).
6. Regulation  
   CDK18’s regulation is achieved through multiple mechanisms that include cyclin binding and phosphorylation by other kinases. A critical regulatory interaction is its binding to cyclin A2, which activates CDK18 by inducing conformational changes in the kinase domain that facilitate substrate access and full catalytic activity. In addition to cyclin A2 binding, CDK18 is also activated by phosphorylation mediated by protein kinase A (PKA). Phosphorylation at specific serine residues, most notably at Ser12, has been shown to significantly enhance the kinase activity of CDK18 even in the absence of cyclin binding; additional phosphorylation sites, such as Ser66 and Ser109, have been identified although their roles appear less pivotal for activation (matsuda2014pctairekinase3cyclindependent pages 10-11, matsuda2014pctairekinase3cyclindependent pages 12-13, pepino2021overviewofpctk3cdk18 pages 1-2).  
   Moreover, CDK18 has been reported to interact with 14-3-3 proteins upon its phosphorylation at one or more of these sites, which may modulate its subcellular localization and stability. Regulatory control of CDK18 also involves feedback mechanisms wherein its activation can influence the stability of cyclin A2, further integrating it into the cell cycle and signal transduction networks (matsuda2014pctairekinase3cyclindependent pages 13-14, pepino2021overviewofpctk3cdk18 pages 17-18). In addition, CDK18 plays a role in replication stress response pathways, wherein its activity is essential for the proper recruitment and retention of checkpoint proteins such as RAD9, RAD17, and TOPBP1 during ATR-mediated signaling (barone2016humancdk18promotes pages 1-1). This multifactorial regulation involving both protein–protein interactions and site-specific phosphorylation events distinguishes CDK18 from many of its classical cell cycle counterparts (pepino2021overviewofpctk3cdk18 pages 11-13).
7. Function  
   CDK18 is predominantly expressed in terminally differentiated, post-mitotic cells, with a particularly high expression in neuronal tissues and oligodendrocytes. Its biological functions are linked primarily to signal transduction processes in these cells rather than to classical regulation of the cell cycle. Functionally, CDK18 has been implicated in the maintenance of genome integrity through its role in the replication stress response. In this capacity, CDK18 promotes the recruitment and phosphorylation of replication checkpoint mediators—such as RAD9, RAD17, and TOPBP1—thereby contributing to ATR activation and stabilization of stalled replication forks. This function is critical for preventing chromosomal abnormalities and the accumulation of DNA damage (barone2016humancdk18promotes pages 1-1, pepino2021overviewofpctk3cdk18 pages 7-9).  
   In addition to its role in genome maintenance, CDK18 exerts influence on cytoskeletal dynamics. By regulating the phosphorylation status of actin-regulating proteins such as cofilin, CDK18 modulates actin polymerization, cell adhesion, and migration. For instance, reduced CDK18 activity has been associated with increased cofilin phosphorylation and consequent accumulation of filamentous actin at the cell periphery, which in turn affects cell motility and invasive behavior (matsuda2014pctairekinase3cyclindependent pages 13-14, pepino2021overviewofpctk3cdk18 pages 7-9).  
   Further, CDK18 has been linked to the differentiation of oligodendrocyte precursor cells via activation of the ERK/MAPK signaling cascade. This role in neural cell differentiation, together with its involvement in the regulation of aquaporin-2 trafficking through interaction with ubiquitin ligase STUB1, suggests additional functions in cell differentiation and intracellular protein trafficking (pepino2021overviewofpctk3cdk18 pages 17-18, pepino2021overviewofpctk3cdk18 pages 19-20).  
   In the context of oncogenic signaling, altered expression of CDK18 has been observed in several cancer types, including breast cancer and glioblastoma, where it contributes to replication stress signaling and influences responses to therapeutic agents such as PARP inhibitors (barone2016humancdk18promotes pages 1-2, pepino2021overviewofpctk3cdk18 pages 19-20). Collectively, these findings indicate that CDK18 functions as a multifaceted kinase involved in genome stability, cytoskeletal reorganization, and cell differentiation, particularly in specialized post-mitotic cell populations (pepino2021overviewofpctk3cdk18 pages 17-18).
8. Other Comments  
   Currently, no CDK18-specific inhibitors have been fully characterized; however, several compounds developed for related CDKs are under investigation for their potential activity against atypical kinases such as CDK18. Inhibitor studies conducted on related PCTAIRE kinases, including insights from inhibitor binding profiles in CDK16, suggest that CDK18 may exhibit a unique sensitivity profile with a limited spectrum of small-molecule engagement (karimbayli2024insightsintothe pages 6-7, pepino2021overviewofpctk3cdk18 pages 4-6).  
   Disease associations for CDK18 include its implication in the modulation of replication stress and genome stability, which are critical processes in cancer biology. Overexpression of CDK18 and copy number gains have been reported in certain breast cancer subtypes, and its involvement in ATR-mediated signaling may contribute to therapy resistance in glioblastoma. In the nervous system, aberrant regulation of CDK18 has been linked to neurodegenerative conditions and altered neuronal functions, with its regulatory effect on tau phosphorylation suggested to play a role in Alzheimer’s disease neuropathology (barone2016humancdk18promotes pages 1-2, pepino2021overviewofpctk3cdk18 pages 13-14).  
   In summary, while extensive structural and biochemical characterizations are available for several related PCTAIRE kinases, CDK18 remains less well studied. Its dual mode of activation—through cyclin binding and PKA-mediated phosphorylation—and its specialized functions in post-mitotic cells render it a promising target for further investigation in both cancer and neurological disorders (pepino2021overviewofpctk3cdk18 pages 14-16).
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