1. Phylogeny  
   Cyclin‐dependent kinase 18 (CDK18), also known as PCTAIRE3 or PCTK3, is a member of the PCTAIRE subfamily of the cyclin‐dependent kinases, which diverged from the classical cell cycle CDKs early in eukaryotic evolution. Its orthologs are found widely in metazoans, and its evolutionary conservation is evident from sequence comparisons that reveal a kinase domain sharing approximately 52–60% identity with related kinases such as CDK16 (PCTAIRE1) and CDK17 (PCTAIRE2) (pepino2021overviewofpctk3cdk18 pages 1-2, cole2009pctkproteinsthe pages 2-4). CDK18 is assigned to the atypical CDK group that does not feature the canonical PSTAIRE motif but instead carries a defining PCTAIRE sequence in its C-helix, a change that is thought to influence its cyclin-binding properties and regulatory interactions (malumbres2014cyclindependentkinases pages 6-7, lazzaro1997characterizationofa pages 32-34). This kinase has been phylogenetically linked to other kinases involved both in cell cycle-independent processes (especially those in terminally differentiated cells) and in key signaling cascades, thereby placing it in a distinct functional and evolutionary branch within the overall human kinome (pepino2021overviewofpctk3cdk18 pages 1-2, karimbayli2024insightsintothe pages 4-6).
2. Reaction Catalyzed  
   CDK18 catalyzes the transfer of a phosphate group from ATP to specific serine/threonine residues on target protein substrates. The reaction can be described by the general equation: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (malumbres2014cyclindependentkinases pages 6-7, barone2016humancdk18promotes pages 1-2).
3. Cofactor Requirements  
   The enzymatic activity of CDK18, like that of other cyclin‐dependent serine/threonine kinases, depends on the presence of divalent metal ions, most notably Mg²⁺, which is required for ATP binding and catalysis (malumbres2014cyclindependentkinases pages 6-7).
4. Substrate Specificity  
   CDK18 functions as a serine/threonine kinase; although its precise consensus substrate motif has not been completely delineated, it is understood to display substrate specificity characteristics shared by many proline‐directed kinases. Available experimental data and proteomic surveys suggest that its substrate preferences resemble those of other CDKs, favoring phosphorylation at serine or threonine residues that are often immediately followed by a proline residue. Additionally, an atlas of substrate specificities for the human serine/threonine kinome indicates that kinases of this family generally target motifs with basic residues surrounding the phosphorylation site; however, the exact amino acid consensus for CDK18 remains less fully characterized (Johnson2023Atlas pages 759-766, pepino2021overviewofpctk3cdk18 pages 2-3).
5. Structure  
   CDK18 comprises a central, highly conserved kinase domain flanked by extended N-terminal and shorter C-terminal regions. The kinase domain contains all the signature motifs found in the CDK family, including the glycine-rich loop involved in ATP binding, the catalytic HRD motif, and the DFG motif essential for coordinating Mg²⁺ and ATP binding (cole2009pctkproteinsthe pages 2-4, malumbres2014cyclindependentkinases pages 6-7). Uniquely, CDK18 harbors a variant motif—namely the PCTAIRE sequence in place of the canonical PSTAIRE—which is critical for its interaction with potential cyclin partners and for dictating conformational dynamics distinct from classical CDKs (pepino2021overviewofpctk3cdk18 pages 2-3, cole2009pctkproteinsthe pages 5-7). Although no experimental crystal structure of CDK18 is currently available, homology modeling based on the resolved structure of CDK16 (PCTAIRE1) (PDB ID: 5G6V) provides insight into its bi-lobal fold with an N-terminal lobe largely composed of β-strands and a C-terminal lobe dominated by α-helices. In this model, structural features such as the activation loop and the C-helix align with those of other active CDKs, while additional unique sequences—especially within the N-terminal extension—may serve as regulatory modules or mediate specific protein-protein interactions (pepino2021overviewofpctk3cdk18 pages 2-3, endicott2013structuralcharacterizationof pages 3-5).
6. Regulation  
   The regulation of CDK18 activity involves multiple layers of control. Key regulatory mechanisms include binding to cyclins—most notably cyclin A2 and potentially cyclin E1—which are traditionally required to induce the conformational changes necessary for full catalytic activity (alonso2021caracterizacióndecdk1418 pages 38-41, pepino2021overviewofpctk3cdk18 pages 11-13). In addition to cyclin binding, phosphorylation plays an important regulatory role; for instance, phosphorylation at serine 12 by protein kinase A (PKA) has been shown to enhance CDK18’s kinase activity independently as well as in conjunction with cyclin binding (alonso2021caracterizacióndecdk1418 pages 38-41, karimbayli2024insightsintothe pages 9-10). CDK18 has also been reported to interact with regulatory proteins such as 14-3-3 and may be subject to further modulation through ubiquitination channels, with components like the ubiquitin ligase STUB1 potentially influencing its stability and localization (alonso2021caracterizacióndecdk1418 pages 35-38, pepino2021overviewofpctk3cdk18 pages 13-14). These post-translational modifications constitute a complex regulatory network that controls the activation state and substrate engagement of CDK18, ensuring appropriate responses in terminally differentiated cells.
7. Function  
   CDK18 is expressed preferentially in terminally differentiated cells, with notable expression in the brain and other post-mitotic tissues, but it is also detected in non-neuronal tissues such as kidney and heart (pepino2021overviewofpctk3cdk18 pages 1-2, cole2009pctkproteinsthe pages 2-4). Functionally, CDK18 is implicated in signal transduction cascades in terminally differentiated cells and may contribute to regulating cell cycle checkpoint responses in non-proliferative contexts. Its activity appears to be linked to modulation of the actin cytoskeleton and vesicular transport processes, including the regulation of aquaporin-2 trafficking via interactions with COPII complex components and regulatory proteins such as STUB1, which functions as an A-kinase anchoring protein (alonso2021caracterizacióndecdk1418 pages 35-38, karimbayli2022dissectingtherole pages 68-71). CDK18 has also been associated with the maintenance of genome stability by participating in the cellular response to replication stress and the ATR-mediated DNA damage response, as evidenced by studies demonstrating increased DNA damage upon CDK18 depletion in cancer cell models (barone2016humancdk18promotes pages 1-2, karimbayli2024insightsintothe pages 18-19). In neuronal cells, CDK18’s capability to phosphorylate substrates such as tau has raised interest in its potential involvement in neurodegenerative disorders, although the detailed substrate spectrum in this compartment remains to be defined (cole2009pctkproteinsthe pages 8-10).
8. Other Comments  
   At present, selective inhibitors that specifically target CDK18 remain poorly characterized; most available small molecule inhibitors tend to be pan-CDK inhibitors that affect a broad spectrum of cyclin-dependent kinases. Some compounds, such as Dabrafenib, have been reported to inhibit related PCTAIRE kinases at clinically relevant concentrations, indicating that CDK18 might be susceptible to similar pharmacological interventions (karimbayli2024insightsintothe pages 6-7, pepino2021overviewofpctk3cdk18 pages 4-6). Disease associations for CDK18 include its involvement in maintaining genomic stability and in signal transduction pathways relevant to cancer biology as well as neurodegenerative diseases like Alzheimer’s disease, where aberrant phosphorylation of tau protein has been noted (cole2009pctkproteinsthe pages 8-10, karimbayli2024insightsintothe pages 13-14). Although detailed reports on specific disease mutations or selective inhibitor efficacies are limited, the available data underscore CDK18’s emerging importance as a potential therapeutic target in oncology and neurology (alonso2021caracterizacióndecdk1418 pages 41-44, barone2016humancdk18promotes pages 12-13).
9. References  
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