1. Phylogeny  
   Cyclin‐dependent kinase 4 (CDK4) belongs to the CMGC group of protein kinases and is a member of the cell‐cycle regulatory subfamily that also includes CDK6. CDK4 and CDK6 are absent in yeasts such as Saccharomyces cerevisiae, and their appearance is associated with the evolution of multicellular organisms. In mammals, CDK4 is found in a distinct evolutionary lineage that diverged from the more ancestral CDC2/CDK1‐like kinases. Phylogenetic analyses indicate that the CDK4/CDK6 subfamily emerged following gene duplication events that occurred after the divergence from unicellular eukaryotes, underscoring their role in the control of the complex cell cycle of metazoans (malumbres2014cyclindependentkinases pages 1-2, malumbres2014cyclindependentkinases pages 2-3). In addition, comparative studies have demonstrated that CDK4 is evolutionarily related to other serine/threonine kinases through conserved structural features in the catalytic domain and regulatory regions. Its grouping with D‐type cyclin partners and its absence in organisms with a simplified cell cycle regulation further support its specialized role in multicellular organisms (pluta2024cyclin‐dependentkinasesmasters pages 3-5, jacques2023roadmaptothe pages 22-25).
2. Reaction Catalyzed  
   CDK4 functions as a serine/threonine protein kinase that catalyzes the transfer of a phosphate group from ATP to specific serine or threonine residues on target proteins. The reaction can be summarized as follows: ATP + [protein substrate] → ADP + [protein substrate]-(phospho-serine/threonine) + H⁺. This phosphorylation event often occurs on substrates bearing a proline immediately following the phosphoacceptor residue, consistent with CDK4’s classification as a proline-directed kinase (baker2012cdk4akey pages 2-3).
3. Cofactor Requirements  
   The catalytic activity of CDK4 is dependent on the presence of divalent cations, with Mg²⁺ being the primary cofactor. The magnesium ion coordinates with ATP in the active site to facilitate the proper orientation and transfer of the phosphate group onto substrate proteins. This requirement is characteristic of most kinases in this family, including those within the CMGC group (baker2012cdk4akey pages 2-3).
4. Substrate Specificity  
   CDK4 exhibits substrate specificity that is mediated by both its catalytic domain and its association with D‐type cyclins. The kinase phosphorylates proteins whose phosphorylation sites generally conform to a serine or threonine followed immediately by a proline (S/T-P motif). In many cases, optimal substrate phosphorylation occurs when a basic residue is present at the P+3 position relative to the phosphoacceptor site, although CDK4 has been shown to phosphorylate substrates even when these full consensus motifs are not strictly met (anders2011asystematicscreen pages 1-2, suryadinata2010controlofcell pages 5-6). Notably, the retinoblastoma protein (RB1) contains several phosphorylation sites that are targeted by CDK4. These sites, including Ser780 and Ser795, are embedded within sequence environments that allow the formation of a consensus motif necessary for efficient phosphate transfer. The specificity is further refined by cyclin D association, which facilitates substrate docking via RXL- or similar linear motifs, and by additional substrate-binding interactions that promote an ordered phosphorylation cycle (johnson2023anatlasof pages 23-26, harper2001cyclindependentkinases pages 12-13).
5. Structure  
   The three-dimensional architecture of CDK4 is organized around a typical kinase fold composed of two lobes. The smaller N-terminal lobe consists of a five-stranded β-sheet structure and a conserved C-helix, while the larger C-terminal lobe is predominantly α-helical. The active site is located in the cleft between these two lobes. In crystal structures of CDK4 bound to cyclin D1, CDK4 adopts an inactive, or “intermediate,” conformation even when its T-loop is phosphorylated on Thr172. Structural studies have revealed that, unlike other CDKs such as CDK2 where cyclin association induces a canonical active conformation characterized by a repositioned C-helix and an open activation loop, the CDK4/cyclin D complex maintains an arrangement that does not allow full substrate access (day2009crystalstructureof pages 1-1, takaki2009thestructureof pages 1-1). The crystal structure shows that the cyclin binds predominantly to the N-lobe and interacts with the C-helix through conserved interfaces. Additionally, a modified loop region in CDK4 (used in crystallographic constructs) has been employed to stabilize the overall structure; however, this engineered modification does not significantly alter the overall domain organization. Key structural features include the ATP-binding site, the glycine-rich P-loop, and the activation segment (T-loop), whose conformation is critical for regulating kinase activity. The distinct inactive conformation observed in CDK4 supports the concept that additional regulatory interactions or substrate binding may be required to fully trigger its enzymatic activity (day2009crystalstructureof pages 1-3, takaki2009thestructureof pages 1-3, wood2018structuralinsightsinto pages 7-8).
6. Regulation  
   CDK4 regulation is achieved through multiple layers of control that include both post-translational modifications and protein–protein interactions. Phosphorylation of the T-loop residue, Thr172, is essential for catalytic activity, although in isolation this modification does not induce the fully active conformation commonly seen in other kinases (baker2022cdk4amaster pages 1-2, takaki2009thestructureof pages 3-3). Binding of D-type cyclins (D1, D2, or D3) is required to form the CDK4/cyclin complex, and this association is integral to both substrate recognition and activity regulation. Endogenous inhibitors such as the INK4 family proteins—most notably p16INK4A—bind to CDK4 and block its association with cyclin D, thereby inhibiting kinase activity by distorting the active site and preventing proper conformational rearrangements (bockstaele2006regulationofcdk4 pages 1-2, baker2012cdk4akey pages 2-3). In addition, members of the CIP/KIP family, such as p21CIP1 and p27KIP1, interact with already formed CDK-cyclin complexes, acting as modulators that can either inhibit or stabilize the enzyme depending on their phosphorylation state. The interplay between these inhibitors, cyclin binding, and T-loop phosphorylation establishes a finely tuned regulatory system, ensuring that CDK4 activity is tightly controlled during the G1 phase of the cell cycle (baker2022cdk4amaster pages 2-4, hallett2017structuralandfunctional pages 30-34).
7. Function  
   CDK4 plays an essential role in cell-cycle progression by mediating the G1 to S phase transition. In complex with D-type cyclins, CDK4 phosphorylates key substrates such as the retinoblastoma family of proteins (including RB1, p107, and p130), which results in the dissociation of E2F transcription factors from their complexes. This leads to the transcriptional activation of genes required for S-phase entry and DNA replication (anders2011asystematicscreen pages 1-2, baker2012cdk4akey pages 2-3). In early G1 phase, cyclin D-CDK4 complexes hypophosphorylate RB1, setting the stage for subsequent hyperphosphorylation by other kinases (such as cyclin E-CDK2) that effectively inactivate RB-mediated repression. Beyond RB phosphorylation, CDK4 also targets other substrates. For instance, it phosphorylates SMAD3 in a cell-cycle-dependent manner, thereby repressing SMAD3’s transcriptional activity, and it has been implicated in phosphorylating transcription factors such as FOXM1, which plays a role in senescence suppression in cancer cells (anders2011asystematicscreen pages 1-2). The integration of divergent mitogenic and antimitogenic signals by CDK4–cyclin D complexes underlies its status as a major integrator of cell cycle control. This functional role is also supported by observations that mutations affecting CDK4, including those that impair inhibitor binding (for example, mutations at residue R24), lead to increased kinase activity and have been associated with oncogenic phenotypes (sheppard2013thecellcycleregulator pages 1-3, malumbres2014cyclindependentkinases pages 5-6).
8. Other Comments  
   CDK4 is a clinically relevant target in oncology due to its pivotal role in cell cycle regulation and the frequent dysregulation of the cyclin D/CDK4/6–RB pathway in various cancers, including melanoma, breast cancer, and renal cell carcinoma (lee2015cdk4inhibitorsan pages 4-6, sager2022therapeuticpotentialof pages 31-34). Several small-molecule inhibitors, such as palbociclib, ribociclib, and abemaciclib, have been developed to selectively inhibit CDK4/6 activity by targeting the ATP-binding site, and these inhibitors have shown efficacy in preclinical and clinical settings (sager2022therapeuticpotentialof pages 7-9, sheppard2013thecellcycleregulator pages 1-3). CDK4 inhibitors have demonstrated the capacity to induce G1 cell-cycle arrest and reduce cell proliferation in tumors that depend on this pathway. In addition to kinase inhibition, strategies targeting the chaperone machinery responsible for stabilizing CDK4, such as the HSP90–CDC37 complex, are being explored to promote CDK4 degradation in cancer cells (bockstaele2006regulationofcdk4 pages 1-2, wood2018structuralinsightsinto pages 10-11). CDK4 mutations, such as those altering the binding interface for inhibitory proteins like p16, can confer resistance to cell-cycle arrest and contribute to tumorigenesis, further emphasizing the therapeutic importance of developing agents that can effectively target aberrant CDK4 activity (baker2012cdk4akey pages 2-3).
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