1. Phylogeny  
   Cyclin-dependent kinase 2 (CDK2) is a member of the cyclin-dependent kinase (CDK) family that is conserved throughout eukaryotes and plays a central role in cell cycle regulation. CDK2 is ubiquitously present in mammals and shares high sequence and structural homology with other cell cycle–regulating kinases such as CDK1 and CDK3, as originally defined in kinome analyses (wood2018structuralinsightsinto pages 1-2, equinet2004studiesonthe pages 47-51). Phylogenetic assessments classify CDK2 within the CMGC group of serine/threonine kinases, an evolutionarily conserved family that emerged early in eukaryotic evolution and is closely related to other cell cycle kinases outlined by Manning et al. (karimbayli2024insightsintothe pages 1-2). Orthologs of CDK2 are present in diverse eukaryotic species, and its evolutionary conservation underscores its essential functions in controlling complex processes such as DNA synthesis and centrosome duplication (equinet2004studiesonthe pages 44-47, pellarin2025cyclindependentproteinkinases pages 7-8).
2. Reaction Catalyzed  
   CDK2 catalyzes the transfer of the γ‐phosphate from ATP to the hydroxyl group of serine or threonine residues on protein substrates. In its catalytic reaction, ATP and a substrate protein with a serine/threonine residue are converted to ADP and a phosphorylated substrate, thereby modulating the function of its protein targets (meschini2011purinebaseddualinhibitors pages 280-282, pellarin2025cyclindependentproteinkinases pages 4-5).
3. Cofactor Requirements  
   The kinase activity of CDK2 depends critically on the presence of divalent metal ions, with Mg²⁺ serving as a necessary cofactor. Mg²⁺ coordinates with ATP within the active site, ensuring proper positioning of the phosphate groups for effective catalysis (meschini2011purinebaseddualinhibitors pages 51-57, wood2018structuralinsightsinto pages 2-3).
4. Substrate Specificity  
   CDK2 is a serine/threonine kinase that phosphorylates substrates involved in cell cycle regulation, and its substrate recognition is influenced by the amino acid context surrounding the phosphoacceptor site. The consensus phosphorylation motif typically requires a serine or threonine residue followed immediately by proline, as frequently observed in CDK substrates such as the retinoblastoma protein (RB) and NPAT; this preference reflects the conserved structural determinants within the kinase domain (pellarin2025cyclindependentproteinkinases pages 15-16, meschini2011purinebaseddualinhibitors pages 29-35). Additional substrate determinants, such as docking motifs (e.g., the RXL motif recognized on the cyclin partner), further contribute to substrate specificity by facilitating proper orientation and binding of target peptides (wood2018structuralinsightsinto pages 9-10, pellarin2025cyclindependentproteinkinases pages 8-9).
5. Structure  
   CDK2 is a small protein kinase of approximately 33 kDa that adopts a conserved bilobal structure comprising an N-terminal lobe predominantly consisting of β-sheets and a larger C-terminal lobe rich in α-helices. The active site is located in the cleft between these two lobes and is characterized by conserved motifs such as the glycine-rich loop, the hinge region, and the activation loop that includes the critical threonine 160 (T160) residue (meschini2011purinebaseddualinhibitors pages 45-51, wood2018structuralinsightsinto pages 4-5). The N-terminal lobe contributes to ATP binding, while the C-terminal lobe contains the catalytic machinery required for phosphoryl transfer and substrate binding. Upon cyclin binding—most notably to cyclin E during the G1/S transition and cyclin A during S phase—conformational changes are induced that reposition the αC-helix and organize the activation loop into an active conformation (pellarin2025cyclindependentproteinkinases pages 2-4, meschini2011purinebaseddualinhibitors pages 57-64). Structural studies have revealed that the binding interface between CDK2 and its cyclin partners is extensive, ensuring both substrate specificity and optimal catalytic activity; this interface is crucial for the proper alignment of key residues that coordinate ATP binding and phosphate transfer (wood2018structuralinsightsinto pages 6-7, pellarin2025cyclindependentproteinkinases pages 49-50).
6. Regulation  
   The regulatory mechanisms governing CDK2 activity are multifaceted and depend primarily on cyclin association, phosphorylation events, and the binding of specific CDK inhibitors (CKIs). CDK2 requires association with cyclin E in early cell cycle phases and with cyclin A in later phases to achieve full activation; cyclin binding induces significant conformational changes that expose the active site for catalysis (equinet2004studiesonthe pages 51-55, pellarin2025cyclindependentproteinkinases pages 4-5). A critical regulatory step involves phosphorylation of threonine 160 in the activation loop by the CDK-activating kinase (CAK), a complex that comprises CDK7, cyclin H, and MAT1; this phosphorylation stabilizes the active conformation of CDK2 (pellarin2025cyclindependentproteinkinases pages 11-12, wood2018structuralinsightsinto pages 10-11). Conversely, inhibitory phosphorylation at residues such as threonine 14 and tyrosine 15 by Wee1 and Myt1 kinases suppresses CDK2 activity, and these inhibitory phosphates are removed by Cdc25 phosphatases to permit full activation (meschini2011purinebaseddualinhibitors pages 51-57, pellarin2025cyclindependentproteinkinases pages 19-20). In addition, binding of endogenous CDK inhibitors from the Cip/Kip family (e.g., p21^Cip1, p27^Kip1, and p57^Kip2) prevents substrate and ATP binding by stabilizing inactive conformations of the CDK2–cyclin complex (pellarin2025cyclindependentproteinkinases pages 7-8, meschini2011purinebaseddualinhibitors pages 35-41).
7. Function  
   CDK2 is a central regulator of the cell cycle whose activity is critical for the G1/S transition and the subsequent progression through S phase and G2 phase. Its activation is essential to initiate DNA synthesis by phosphorylating key substrates such as the retinoblastoma protein (RB), which in turn releases E2F transcription factors that drive the transcription of genes required for DNA replication (pellarin2025cyclindependentproteinkinases pages 50-51, meschini2011purinebaseddualinhibitors pages 57-64). In addition to its canonical role in cell cycle progression, CDK2 is involved in diverse processes including centrosome duplication, homologous recombination–dependent DNA repair, and the modulation of transcription by phosphorylating factors such as NPAT, which in turn activates histone gene transcription during S phase (equinet2004studiesonthe pages 47-51, pellarin2025cyclindependentproteinkinases pages 6-7). In embryonic stem cells, CDK2 contributes to maintaining the delicate balance between proliferation, cell death, and DNA repair, thereby influencing cell fate decisions and pluripotency (pellarin2025cyclindependentproteinkinases pages 8-9, meschini2011purinebaseddualinhibitors pages 64-69). CDK2 also phosphorylates proteins implicated in the DNA damage response, including BRCA2 and NBN, thereby integrating cell cycle regulation with genome stability mechanisms (pellarin2025cyclindependentproteinkinases pages 19-20, meschini2011purinebaseddualinhibitors pages 282-284). Its activity is tightly upregulated during S phase and G2 as it prepares the cell for mitotic entry by controlling the activation of cyclin B/CDK1 complexes (pellarin2025cyclindependentproteinkinases pages 51-51, equinet2004studiesonthe pages 55-58).
8. Other Comments  
   Several experimental and small molecule inhibitors targeting CDK2 have been developed owing to its critical role in promoting cell proliferation, particularly in cancer cells where CDK2 is frequently dysregulated. Inhibitors targeting the ATP-binding site have been designed to compete with ATP and disrupt kinase activity, and preclinical studies have evaluated dual inhibitors of CDK2 and related kinases such as CDK7 (meschini2011purinebaseddualinhibitors pages 277-280, meschini2011purinebaseddualinhibitors pages 282-284). CDK2 dysregulation is associated with a broad spectrum of cancers including breast, glioblastoma, and colorectal cancers, as well as with defects in meiotic processes leading to infertility (pellarin2025cyclindependentproteinkinases pages 20-20, colas2020cyclindependentkinasesand pages 1-2). Beyond oncogenesis, CDK2 has been linked to the regulation of transcriptional programs through phosphorylation of factors such as MYC and EZH2, which modulate epigenetic gene silencing via H3K27me3 maintenance (pellarin2025cyclindependentproteinkinases pages 50-51, meschini2011purinebaseddualinhibitors pages 45-51). Notable mutations in regulatory regions or phosphorylation sites—such as those affecting the T160 activation loop—have the potential to alter its kinase activity and contribute to disease; however, specific pathogenic mutations in CDK2 are not as frequently reported as alterations in its regulatory partners (meschini2011purinebaseddualinhibitors pages 64-69, karimbayli2024insightsintothe pages 17-18). Overall, the therapeutic potential of targeting CDK2 in cancer and other proliferative disorders continues to be a significant focus of research, particularly as more selective inhibitors are developed that exploit the highly conserved structural and catalytic features of this kinase (wood2018structuralinsightsinto pages 8-9, pellarin2025cyclindependentproteinkinases pages 18-19).
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