1. Phylogeny  
   Cyclin‐dependent kinase 6 (CDK6) is a member of the cell cycle CDK subfamily that is highly conserved across metazoans. CDK6 is phylogenetically related to other G1 phase kinases such as CDK4, and its origin can be traced back to the common eukaryotic ancestor along with other core CDKs involved in cell cycle regulation (colas2020cyclindependentkinasesand pages 1-2, pluta2024cyclin‐dependentkinasesmasters pages 23-25). Within the human kinome, CDK6 is classified in the cell cycle regulatory group and shares structural and functional characteristics with CDK4, yet it exhibits distinct tissue‐specific expression patterns and substrate specificities. Comparative genomic analyses have shown that CDK6 and its paralog CDK4 evolved by gene duplication in higher eukaryotes, and both kinases have maintained structural homology with the ancestral CDK proteins originally described in yeast (harper2001cyclindependentkinases pages 2-4, colas2020cyclindependentkinasesand pages 1-2).
2. Reaction Catalyzed  
   CDK6 catalyzes the transfer of a phosphate group from ATP to specific serine/threonine residues on target substrate proteins. The reaction can be summarized as follows:  
     ATP + [protein] – (L‑serine or L‑threonine) → ADP + [protein] – (L‑serine/threonine‑phosphate) + H⁺  
   This catalytic reaction is fundamental for modifying the activity and regulatory functions of key substrates such as retinoblastoma protein (pRB/RB1) and nucleophosmin (NPM1) (OpenTargets Search: -CDK6).
3. Cofactor Requirements  
   The kinase activity of CDK6 is dependent on the presence of Mg²⁺ ions, which function as essential cofactors by coordinating the phosphate groups of ATP during the catalytic reaction. Mg²⁺ is required for the proper binding of ATP to the conserved kinase domain, thereby facilitating efficient catalysis (knockaert2002pharmacologicalinhibitorsof pages 2-3).
4. Substrate Specificity  
   CDK6 phosphorylates serine/threonine residues on target proteins that are crucial for cell cycle progression and differentiation. One of the primary substrates is the retinoblastoma protein (RB1), whose phosphorylation leads to conformational changes that release E2F transcription factors and promote the G1/S transition. In addition to pRB/RB1, CDK6 is known to phosphorylate nucleophosmin (NPM1) as well as additional substrates involved in differentiation and cytoskeletal reorganization (OpenTargets Search: -CDK6). Although a detailed consensus substrate motif for CDK6 is not explicitly defined in the available data, studies of the related G1 kinases suggest that substrate recognition by CDK6 is dependent upon the interaction with its D-type cyclin partners, which modulate the enzyme’s substrate binding groove and may impose preferences for specific amino acid sequences surrounding the phosphorylated residue (law2015cyclindependentkinaseinhibitors pages 2-3, pellarin2025cyclindependentproteinkinases pages 2-4).
5. Structure  
   CDK6 possesses a canonical kinase domain that is characteristic of the serine/threonine protein kinases. The central kinase domain features a two-lobed structure: an N-terminal lobe, which is predominantly composed of β-sheets and includes the conserved PSTAIRE helix, and a larger C-terminal lobe composed largely of α-helices that contain the catalytic core and substrate-binding regions (endicott2013structuralcharacterizationof pages 1-2, wood2018structuralinsightsinto pages 19-20). Key structural elements include the activation loop (T-loop), whose phosphorylation is necessary for achieving full catalytic activity, the glycine-rich loop that contributes to ATP binding, and the conserved DFG motif that coordinates Mg²⁺ and ATP (hallett2017structuralandfunctional pages 27-30, wood2018structuralinsightsinto pages 19-20). Furthermore, binding of a D-type cyclin to CDK6 induces a conformational re-arrangement, particularly repositioning the C-helix from an “out” to an “in” conformation, which is crucial for aligning catalytic residues and substrate recognition. Unique to CDK6 in comparison with other family members, differences in the structure of its substrate-binding region and in its interactions with regulatory proteins (e.g., chaperones such as Hsp90/Cdc37) have been noted and contribute to its distinct functional profile (hallett2017structuralandfunctional pages 38-42, wood2018structuralinsightsinto pages 7-8).
6. Regulation  
   Full activation of CDK6 is dependent on its association with D-type cyclins (cyclin D1, D2, or D3), which under mitogenic stimuli bind to CDK6 during the G1 phase to form an active complex that phosphorylates downstream substrates. In addition, CDK6 activity is regulated post-translationally by phosphorylation events. Activation loop phosphorylation, typically mediated by CDK-activating kinases (CAK) such as CDK7 in complex with cyclin H and MAT1, is required to enhance the catalytic efficiency of CDK6 (hallett2017structuralandfunctional pages 20-24, law2015cyclindependentkinaseinhibitors pages 1-2). Conversely, CDK6 can be negatively regulated by inhibitory phosphorylation events mediated by kinases such as Wee1 and Myt1, although direct inhibitory phosphorylation events on CDK6 are less extensively characterized than those on other CDKs (suryadinata2010controlofcell pages 1-3). Moreover, CDK6 is subject to regulation by cyclin-dependent kinase inhibitors (CKIs) from the INK4 family (e.g., p16INK4a) and the CIP/KIP family (e.g., p21CIP1, p27KIP1). Binding of these inhibitors can block the association of CDK6 with D-type cyclins or displace preformed active complexes, thereby preventing substrate phosphorylation and cell cycle progression (knockaert2002pharmacologicalinhibitorsof pages 3-4, schmitz2016cyclindependentkinasesas pages 1-2). Additionally, chaperone-mediated regulation involving Hsp90 and Cdc37 contributes to the proper folding and stabilization of CDK6 prior to cyclin binding, distinguishing its regulation from that of its close homolog CDK4 (wood2018structuralinsightsinto pages 7-8, hallett2017structuralandfunctional pages 38-42).
7. Function  
   CDK6 plays a critical role in the control of the cell cycle, particularly in governing the G1/S transition. By phosphorylating the retinoblastoma protein (pRB/RB1), CDK6 facilitates the release of E2F transcription factors, which in turn initiate the transcription of genes required for DNA synthesis and S phase entry (OpenTargets Search: -CDK6, suryadinata2010controlofcell pages 9-10). In addition to its quintessential role in cell cycle progression, CDK6 is involved in several aspects of cellular differentiation and proliferation. It is essential for the proliferation of specific cell types such as hematopoietic cells, thymocytes, and beta-cells in the pancreatic islets, and it is required for the production of newborn neurons in neurogenic regions such as the dentate gyrus and the subventricular zone (OpenTargets Search: -CDK6). CDK6 also contributes to the initiation and maintenance of cell cycle exit during differentiation in various tissues by modulating gene expression patterns and cytoskeletal organization. For example, in astrocytes, activation of CDK6 is associated with reorganization of the actin cytoskeleton and enhanced cellular motility, while in myeloid cells, CDK6 can prevent differentiation by interfering with transcription factor RUNX1 (OpenTargets Search: -CDK6, pluta2024cyclin‐dependentkinasesmasters pages 45-46). Furthermore, CDK6 has been implicated in delaying senescence and modulating the transcription of genes that influence cell fate decisions during differentiation (hallett2017structuralandfunctional pages 223-226). Through its kinase-dependent and kinase-independent functions, CDK6 serves as an integrator of proliferative signals and differentiation cues, thereby playing a pivotal role in both normal physiology and oncogenesis (law2015cyclindependentkinaseinhibitors pages 2-3, hydbring2016noncanonicalfunctionsof pages 1-3).
8. Other Comments  
   CDK6 is a well-established target for anticancer therapy, with several small-molecule inhibitors, such as palbociclib, ribociclib, and abemaciclib, currently approved for the treatment of hormone receptor-positive breast cancer (law2015cyclindependentkinaseinhibitors pages 2-3, milletti2023cyclers’kinasesin pages 2-3). These inhibitors typically function by competing with ATP for binding to the conserved kinase domain of CDK6, thereby reducing the phosphorylation of key substrates like pRB and halting cell cycle progression. Dysregulation of CDK6, whether by overexpression, gene amplification, or loss of inhibitory signals such as those provided by p16INK4a, has been implicated in a variety of malignancies including hematological cancers, breast cancer, melanoma, and gliomas (OpenTargets Search: -CDK6, pellarin2025cyclindependentproteinkinases pages 53-54). Although specific mutation details in CDK6 itself are less frequently reported relative to alterations in its regulatory network, changes affecting its expression or interactions with cyclins and CKIs have significant impacts on cellular proliferation and differentiation outcomes (dallacqua2016cdk6controlsplatinum pages 13-16). In addition to its role in cell proliferation, CDK6 has been associated with non-canonical functions, such as the modulation of transcription factor activity and the regulation of cytoskeletal dynamics during cell differentiation (OpenTargets Search: -CDK6, pluta2024cyclin‐dependentkinasesmasters pages 5-7). These multifaceted roles underscore CDK6’s significance as a master regulator of cell cycle progression and cellular differentiation, making it a prominent focus for further therapeutic intervention (schmitz2016cyclindependentkinasesas pages 5-7, wood2018structuralinsightsinto pages 10-11).
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