1. Phylogeny  
   Cyclin‐dependent kinase 1 (CDK1), also known by its synonyms CDC2, CDC28A, and p34 protein kinase, belongs to the core group of cell‐cycle regulatory kinases that emerged early in eukaryotic evolution. CDK1 is evolutionarily highly conserved and can be traced back to the common ancestral CDK present in yeast species such as Saccharomyces cerevisiae (where the homolog is CDC28) and Schizosaccharomyces pombe (where it is known as CDC2) as well as in invertebrates and vertebrates, including mammals. Structurally and functionally, CDK1 is closely related to CDK2, sharing approximately 65% sequence identity, yet it is uniquely indispensable for cell-cycle progression across eukaryotes. It occupies the cell‐cycle CDK subgroup that also includes CDK2, CDK4, and CDK6, and is part of an evolutionarily conserved regulatory network that integrates cyclin binding and phosphorylation events fundamental to mitosis (brown2015cdk1structuresreveal pages 1-2, ding2020therolesof pages 5-7, korolchuk2018structuralandfunctional pages 31-35).
2. Reaction Catalyzed  
   CDK1 catalyzes an ATP‐dependent phosphorylation reaction that transfers the γ‐phosphate from ATP to serine or threonine residues on substrate proteins. The reaction can be summarized as: ATP + [protein]–(L‑serine/threonine) → ADP + [protein]–(L‑serine/threonine)-phosphate + H⁺. This phosphorylation alters the conformation, activity, or interaction pattern of the substrate proteins, thereby regulating critical processes such as centrosome separation, nuclear envelope breakdown, and chromatin condensation during the cell cycle (brown2015cdk1structuresreveal pages 1-2, massacci2023thecyclindependentkinase pages 2-3).
3. Cofactor Requirements  
   The kinase activity of CDK1 requires ATP as the phosphate donor and typically depends on Mg²⁺ as a critical metal ion cofactor to correctly coordinate the ATP molecule within its catalytic cleft. These cofactors are essential for the proper alignment of catalytic residues and for facilitating the phosphoryl transfer reaction. In addition to magnesium ions, the activation of CDK1 is also dependent on association with cyclin proteins, which while not a cofactor in the classical sense, is essential for its conformational activation and substrate recognition (pellarin2025cyclindependentproteinkinases pages 2-4, wang2023functionsofinteractions pages 17-23).
4. Substrate Specificity  
   CDK1 is a serine/threonine kinase that phosphorylates a broad spectrum of proteins, with its substrate specificity largely dictated by the consensus sequence motif “S/T-P” frequently extended to include additional basic residues downstream, forming a motif such as S/T-P-X-K/R. This consensus motif is recognized within numerous substrates that orchestrate mitotic events. CDK1 phosphorylates a wide array of substrates, including core regulators of mitosis such as lamin proteins (LMNA, LMNB), components of the spindle apparatus, nuclear envelope proteins, and regulators of centrosome dynamics. Other known substrates include transcription factors (e.g., FOXO1), apoptosis regulators (e.g., Bcl-xL/BCL2L1), and proteins involved in DNA damage repair (e.g., BRCA2). The association with different cyclins (A and B families) can modulate the substrate specificity further; for example, complex formation with cyclin B is critical for targeting proteins that mediate nuclear envelope breakdown and chromosome condensation, while cyclin A binding extends activity into late G2 and early mitosis (ding2020therolesof pages 5-7, mcgrath2016cksandspeedy pages 9-12, peyressatre2015targetingcyclindependentkinases pages 1-4).
5. Structure  
   CDK1 displays a canonical protein kinase fold that comprises a smaller N-terminal lobe predominantly formed by β-sheets and a larger C-terminal lobe rich in α-helices. A key structural feature is the PSTAIRE helix located in the N-terminal domain, which is critical for cyclin binding and subsequent activation; this motif is highly conserved among CDKs. Also central to its regulation is the activation loop (T-loop) whose phosphorylation at a conserved threonine residue (analogous to Thr161 in many kinases) is necessary for full enzymatic activity. The inactive, monomeric form of CDK1 is characterized by a “C-helix out” conformation, and cyclin binding induces a conformational rearrangement that repositions the C-helix to form an active catalytic site. Structural studies have indicated that the CDK1–cyclin complexes, while sharing the overall bilobal kinase architecture with other CDKs such as CDK2, exhibit distinct interfacial surfaces and activation kinetics that contribute to differences in substrate specificity and thermal stability (korolchuk2018structuralandfunctional pages 23-26, korolchuk2018structuralandfunctional pages 62-68, pluta2024cyclin‐dependentkinasesmasters pages 12-14).
6. Regulation  
   Regulation of CDK1 is achieved via multiple converging mechanisms that ensure precise control over mitotic entry and progression. First, cyclin binding is essential for conformational activation; CDK1 associates primarily with cyclins A and B, whose expression levels oscillate throughout the cell cycle, peaking during the G2/M phases. Second, phosphorylation events play central roles: activating phosphorylation of the T-loop (e.g., Thr161, by CDK-activating kinase, CAK complex) is required for full activation, whereas inhibitory phosphorylation at Thr14 and Tyr15—mediated by kinases such as Wee1 and Myt1—prevents premature activation by blocking ATP binding. The removal of these inhibitory phosphorylations by CDC25 phosphatases initiates mitotic entry. Additional layers of regulation include binding of regulatory subunits such as Cks proteins that facilitate multisite phosphorylation of substrates and modulate the kinase’s activity. The precise temporal control by these phosphorylation cascades, along with cyclin degradation via the ubiquitin–proteasome system during mitotic exit, ensures that CDK1 activity is tightly confined to appropriate stages of the cell cycle (brown2015cdk1structuresreveal pages 1-2, ding2020therolesof pages 5-7, wijnen2021cyclindependentkinase1 pages 2-4, poulainUnknownyearinvestigatingp21mediateddynamic pages 147-150).
7. Function  
   CDK1 functions as the master regulator of the eukaryotic cell cycle. Its primary role is to govern the G2/M transition and to coordinate the events of mitosis. Upon activation, CDK1 phosphorylates a plethora of substrates involved in initiating centrosome separation, nuclear envelope breakdown, chromatin condensation, and spindle assembly. These coordinated phosphorylation events enable timely progression into mitosis. In addition, CDK1 is implicated in other cellular processes beyond its classical role in mitosis. For example, it is involved in DNA damage response pathways by enforcing the G2 checkpoint through inhibitory phosphorylations that halt cell cycle progression until repairs are complete. In proliferating cells, CDK1 also phosphorylates transcription factors and proteins associated with apoptosis, thereby linking cell cycle dynamics to mechanisms that control cell survival. In early embryogenesis, CDK1 activity is essential, with knockout models demonstrating embryonic lethality due to defective cell division. Dysregulation of CDK1 activity is strongly associated with oncogenesis, as its aberrant activation can result in uncontrolled proliferation and genomic instability, making it an attractive target for cancer therapeutics (ding2020therolesof pages 5-7, garciareyes2018theemergingrole pages 3-4, pluta2024investigatingtherole pages 288-291, shah2020cdksfamilya pages 3-4).
8. Other Comments  
   Given its central role in cell cycle regulation and its involvement in critical mitotic events, CDK1 is a major target of pharmacological inhibitors currently under investigation for cancer therapy. Inhibitors such as RO-3306 have been designed to specifically target the ATP-binding site of CDK1 and have shown promise in preclinical studies by inducing cell-cycle arrest and promoting apoptosis in tumor cells. Overexpression or deregulation of CDK1 has been documented in various cancers, where it contributes to uncontrolled proliferation and resistance to DNA damage–induced apoptosis. Although naturally occurring mutations in CDK1 are less frequently reported compared to other CDKs, disruptions in its regulatory network, including aberrant cyclin expression or faulty phosphorylation control, can dramatically impact cell-cycle fidelity and genomic stability. Current research continues to unravel the broader roles of CDK1 in transcriptional regulation and even in non-mitotic cellular processes such as mitochondrial function, expanding its relevance from a simple cell-cycle driver to a multifunctional regulatory kinase (massacci2023thecyclindependentkinase pages 1-2, massacci2023thecyclindependentkinase pages 9-9, peyressatre2015targetingcyclindependentkinases pages 6-8, alrawi2023cyclinaand pages 35-37).
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