1. Phylogeny  
   CDK6 belongs to the cyclin‐dependent kinase (CDK) family, a group of serine/threonine kinases that evolved from a single ancestral kinase present in lower eukaryotes and expanded in metazoans into multiple members with specialized roles. In particular, CDK6 is a member of the CDK4/6 subfamily, which is found exclusively in eumetazoans and is evolutionarily more relevant for controlling cell cycle entry in higher organisms (asghar2015thehistoryand pages 1-2, malumbres2014cyclindependentkinases pages 2-3). Orthologs of CDK6 are known to exist in all mammalian species, and phylogenetic analyses using the complete human kinome have assigned CDK6 to the CMGC group, sharing significant overall sequence and functional similarity with other cell-cycle kinases such as CDK4 and CDK1 (fassl2022cdk4andcdk6 pages 1-3, malumbres2005mammaliancyclindependentkinases pages 1-2). The evolutionary expansion of the CDK family from yeast to man further underlines the specialized regulatory functions that CDK6 has acquired, notably in the transition through the G1 phase and in the coordination of differentiation-specific processes (asghar2015thehistoryand pages 1-2, malumbres2014cyclindependentkinases pages 8-9).
2. Reaction Catalyzed  
   CDK6 catalyzes the ATP-dependent phosphorylation of serine/threonine residues on protein substrates. The canonical reaction can be described as: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (huwe2003smallmoleculesas pages 1-3). This enzymatic process is central to cell cycle control since phosphorylation of substrates such as the retinoblastoma protein (Rb) alleviates transcriptional repression and permits cell cycle progression (asghar2015thehistoryand pages 9-10).
3. Cofactor Requirements  
   The kinase activity of CDK6 requires the presence of divalent metal ions, with Mg²⁺ serving as a critical cofactor for the binding of ATP, which is necessary for catalysis. The typical dependence on Mg²⁺ is consistent with the requirements for the entire family of serine/threonine kinases (huwe2003smallmoleculesas pages 1-3).
4. Substrate Specificity  
   CDK6 exhibits substrate specificity that is largely determined by its association with D-type cyclins, which modulate its affinity for substrates and help define the context in which it phosphorylates proteins. Its best‐characterized substrates include Rb, as well as other proteins such as NPM1, where the phosphorylation events occur predominantly on serine and threonine residues adjacent to proline residues. Although no exclusive consensus motif is provided in the current context, the overall serine/threonine–proline preference is typical for CDKs (asghar2015thehistoryand pages 9-10, huwe2003smallmoleculesas pages 1-3). Additional substrate specificity is conferred by the cyclin partner, which may alter the conformational landscape of the active site to favor phosphorylation of particular substrates involved in cell cycle regulation (fassl2022cdk4andcdk6 pages 3-4, malumbres2014cyclindependentkinases pages 3-5).
5. Structure  
   CDK6 displays the canonical bilobal structure characteristic of protein kinases, with an N-terminal lobe that is rich in β-sheets and contains a glycine-rich loop critical for ATP binding, and a larger C-terminal lobe composed mainly of α-helices that houses the catalytic machinery. The structures obtained by homology modeling and experimental crystallography of related kinases (e.g., CDK2 and structures of CDK6 bound to viral cyclins) suggest that CDK6 contains key structural features such as the highly conserved ATP-binding pocket, an activation loop that is subject to phosphorylation, and a C-helix whose orientation is critical for catalytic activity (ferrer2006structuralbasisfor pages 3-4, wood2018structuralinsightsinto pages 1-2). Furthermore, the binding of D-type cyclins to CDK6 significantly reorganizes its conformation by stabilizing the active site and aligning the catalytic residues for phosphotransfer. Unique structural aspects of CDK6, when compared with closely related kinases such as CDK4, include differences in the solvent exposure of the C-terminal lobe and distinct interfaces for binding specific inhibitors and regulatory proteins like p16^INK4A (fassl2022cdk4andcdk6 pages 1-3, ferrer2006structuralbasisfor pages 5-6). These features provide both the functional flexibility necessary for its role in cell proliferation and opportunities for the development of selective therapeutic inhibitors.
6. Regulation  
   The activity of CDK6 is tightly regulated by multiple mechanisms that ensure proper cell cycle progression and cellular differentiation. First, the binding of D-type cyclins is essential for CDK6 activation; these cyclins are induced by mitogenic signals and form holoenzyme complexes with CDK6 during the G1 phase (asghar2015thehistoryand pages 1-2, fassl2022cdk4andcdk6 pages 1-3). Additionally, the kinase is subject to activation loop phosphorylation by CDK-activating kinases (CAK), which further enhances its catalytic activity (malumbres2014cyclindependentkinases pages 5-6). CDK6 is negatively regulated by the binding of INK4 family inhibitors, such as p16^INK4A, which block cyclin association and induce conformational changes that inhibit ATP binding (sausville2002complexitiesinthe pages 1-2, sielecki2000cyclindependentkinaseinhibitors pages 5-6). Moreover, members of the Cip/Kip family—p21^Cip1 and p27^Kip1—can bind to preformed cyclin D–CDK6 complexes, affecting substrate recruitment and, in some instances, serving as both inhibitors and assembly factors depending on their phosphorylation state (fassl2022cdk4andcdk6 pages 3-4, peyressatre2015targetingcyclindependentkinases pages 27-30). These multiple layers of regulation ensure that CDK6 activity is coordinated with the cell’s proliferative status and differentiation cues (asghar2015thehistoryand pages 9-10, malumbres2011physiologicalrelevanceof pages 3-4).
7. Function  
   CDK6 plays several critical roles in cellular processes, primarily related to the control of the cell cycle and differentiation. As a serine/threonine kinase, CDK6 promotes the G1/S transition by phosphorylating key substrates such as the retinoblastoma protein (Rb), leading to the release of E2F transcription factors that trigger the expression of genes required for DNA replication (asghar2015thehistoryand pages 9-10, suryadinata2010controlofcell pages 3-4). Beyond its canonical role in cell cycle progression, CDK6 is involved in various tissue-specific processes. It is essential for the proliferation of hematopoietic cells, erythroid progenitors, beta-cells in pancreatic islets, and neuronal progenitors in the dentate gyrus of the hippocampus and the subventricular zone (information section; sherr2016targetingcdk4and pages 1-2). In the context of differentiation, CDK6 participates not only in initiating cell cycle exit but also in maintaining specific differentiation programs by modulating gene expression and cytoskeletal organization, for instance in astrocytes where changes in the actin cytoskeleton and enhanced cellular motility are observed (information section; malumbres2014cyclindependentkinases pages 6-7). CDK6 also interacts with, and in some cases negatively regulates, transcription factors such as RUNX1, thereby preventing myeloid differentiation while promoting the proliferation of normal progenitor cells (information section; malumbres2014cyclindependentkinases pages 8-9). These context-dependent roles underscore the importance of CDK6 in both promoting proliferation when required and in orchestrating appropriate cell cycle exit during differentiation.
8. Other Comments  
   CDK6 is a prominent target in oncology due to its frequent dysregulation in various cancers, where its overexpression or hyperactivation leads to uncontrolled cell proliferation (asghar2015thehistoryand pages 9-10, peyressatre2015targetingcyclindependentkinases pages 15-17). Selective inhibitors such as palbociclib, ribociclib, and abemaciclib are clinically approved agents that target the ATP-binding pocket of CDK6 to prevent Rb phosphorylation and induce G1 phase arrest (sherr2016targetingcdk4and pages 1-2, tadesse2018cyclindependentkinase2 pages 18-23). In addition to cancer, CDK6 has been implicated in other physiological contexts such as thymocyte development and the regulation of cell senescence, as well as in the organization of the centrosome during the cell cycle (information section; suryadinata2010controlofcell pages 9-10). The structural features unique to CDK6 relative to other CDKs provide opportunities for the rational design of inhibitors that exhibit high selectivity, thereby minimizing off-target effects (wood2018structuralinsightsinto pages 6-7, ferrer2006structuralbasisfor pages 5-6). Moreover, the dual role of CDK6 in both cell cycle promotion and transcriptional regulation highlights its potential as a multifaceted therapeutic target (malumbres2014cyclindependentkinases pages 8-9, peyressatre2015targetingcyclindependentkinases pages 6-8).
9. References  
   asghar2015thehistoryand pages 1-2; asghar2015thehistoryand pages 9-10; fassl2022cdk4andcdk6 pages 1-3; fassl2022cdk4andcdk6 pages 3-4; huwe2003smallmoleculesas pages 1-3; malumbres2014cyclindependentkinases pages 2-3; malumbres2014cyclindependentkinases pages 3-5; peyressatre2015targetingcyclindependentkinases pages 1-4; peyressatre2015targetingcyclindependentkinases pages 27-30; sausville2002complexitiesinthe pages 1-2; sherr2016targetingcdk4and pages 1-2; sielecki2000cyclindependentkinaseinhibitors pages 5-6; tadesse2018cyclindependentkinase2 pages 4-8; wood2018structuralinsightsinto pages 1-2; ding2020therolesof pages 1-3; ding2020therolesof pages 5-7; ding2020therolesof pages 7-8; ferrer2006structuralbasisfor pages 3-4; ferrer2006structuralbasisfor pages 4-5; ferrer2006structuralbasisfor pages 5-6; huwe2003smallmoleculesas pages 3-4; knockaert2002pharmacologicalinhibitorsof pages 3-4; knockaert2002pharmacologicalinhibitorsof pages 4-5; lolli2005cak—cyclindependentactivatingkinase pages 6-6; malumbres2005mammaliancyclindependentkinases pages 1-2; malumbres2005mammaliancyclindependentkinases pages 3-4; malumbres2005mammaliancyclindependentkinases pages 6-7; malumbres2011physiologicalrelevanceof pages 3-4; malumbres2014cyclindependentkinases pages 1-2; malumbres2014cyclindependentkinases pages 5-6; malumbres2014cyclindependentkinases pages 6-7; malumbres2014cyclindependentkinases pages 8-9; peyressatre2015targetingcyclindependentkinases pages 15-17; peyressatre2015targetingcyclindependentkinases pages 4-6; peyressatre2015targetingcyclindependentkinases pages 6-8; sielecki2000cyclindependentkinaseinhibitors pages 1-2; suryadinata2010controlofcell pages 3-4; suryadinata2010controlofcell pages 9-10; tadesse2018cyclindependentkinase2 pages 18-23; wang2017themetabolicfunction pages 1-3; wood2018structuralinsightsinto pages 17-18; wood2018structuralinsightsinto pages 18-19; wood2018structuralinsightsinto pages 2-3; wood2018structuralinsightsinto pages 20-20; wood2018structuralinsightsinto pages 6-7; wood2018structuralinsightsinto pages 7-8; fischer2003cyclindependentkinaseinhibitors pages 1-3; knockaert2002pharmacologicalinhibitorsof pages 1-2; lolli2005cak—cyclindependentactivatingkinase pages 4-5; malumbres2005mammaliancyclindependentkinases pages 2-3.

References

1. (asghar2015thehistoryand pages 1-2): Uzma Asghar, Agnieszka K. Witkiewicz, Nicholas C. Turner, and Erik S. Knudsen. The history and future of targeting cyclin-dependent kinases in cancer therapy. Nature Reviews Drug Discovery, 14:130-146, Jan 2015. URL: https://doi.org/10.1038/nrd4504, doi:10.1038/nrd4504. This article has 1892 citations and is from a highest quality peer-reviewed journal.
2. (asghar2015thehistoryand pages 9-10): Uzma Asghar, Agnieszka K. Witkiewicz, Nicholas C. Turner, and Erik S. Knudsen. The history and future of targeting cyclin-dependent kinases in cancer therapy. Nature Reviews Drug Discovery, 14:130-146, Jan 2015. URL: https://doi.org/10.1038/nrd4504, doi:10.1038/nrd4504. This article has 1892 citations and is from a highest quality peer-reviewed journal.
3. (fassl2022cdk4andcdk6 pages 1-3): Anne Fassl, Yan Geng, and Piotr Sicinski. Cdk4 and cdk6 kinases: from basic science to cancer therapy. Science, Jan 2022. URL: https://doi.org/10.1126/science.abc1495, doi:10.1126/science.abc1495. This article has 324 citations and is from a highest quality peer-reviewed journal.
4. (fassl2022cdk4andcdk6 pages 3-4): Anne Fassl, Yan Geng, and Piotr Sicinski. Cdk4 and cdk6 kinases: from basic science to cancer therapy. Science, Jan 2022. URL: https://doi.org/10.1126/science.abc1495, doi:10.1126/science.abc1495. This article has 324 citations and is from a highest quality peer-reviewed journal.
5. (huwe2003smallmoleculesas pages 1-3): Axel Huwe, Ralph Mazitschek, and Athanassios Giannis. Small molecules as inhibitors of cyclin-dependent kinases. Angewandte Chemie, 42 19:2122-38, May 2003. URL: https://doi.org/10.1002/anie.200200540, doi:10.1002/anie.200200540. This article has 235 citations and is from a highest quality peer-reviewed journal.
6. (malumbres2014cyclindependentkinases pages 2-3): Marcos Malumbres. Cyclin-dependent kinases. Genome Biology, 15:122-122, Jun 2014. URL: https://doi.org/10.1186/gb4184, doi:10.1186/gb4184. This article has 1882 citations and is from a highest quality peer-reviewed journal.
7. (malumbres2014cyclindependentkinases pages 3-5): Marcos Malumbres. Cyclin-dependent kinases. Genome Biology, 15:122-122, Jun 2014. URL: https://doi.org/10.1186/gb4184, doi:10.1186/gb4184. This article has 1882 citations and is from a highest quality peer-reviewed journal.
8. (peyressatre2015targetingcyclindependentkinases pages 1-4): Marion Peyressatre, Camille Prével, Morgan Pellerano, and May Morris. Targeting cyclin-dependent kinases in human cancers: from small molecules to peptide inhibitors. Cancers, 7:179-237, Jan 2015. URL: https://doi.org/10.3390/cancers7010179, doi:10.3390/cancers7010179. This article has 402 citations and is from a peer-reviewed journal.
9. (peyressatre2015targetingcyclindependentkinases pages 27-30): Marion Peyressatre, Camille Prével, Morgan Pellerano, and May Morris. Targeting cyclin-dependent kinases in human cancers: from small molecules to peptide inhibitors. Cancers, 7:179-237, Jan 2015. URL: https://doi.org/10.3390/cancers7010179, doi:10.3390/cancers7010179. This article has 402 citations and is from a peer-reviewed journal.
10. (sausville2002complexitiesinthe pages 1-2): Edward A. Sausville. Complexities in the development of cyclin-dependent kinase inhibitor drugs. Trends in Molecular Medicine, 8:S32-S37, Apr 2002. URL: https://doi.org/10.1016/s1471-4914(02)02308-0, doi:10.1016/s1471-4914(02)02308-0. This article has 239 citations and is from a domain leading peer-reviewed journal.
11. (sherr2016targetingcdk4and pages 1-2): Charles J. Sherr, David Beach, and Geoffrey I. Shapiro. Targeting cdk4 and cdk6: from discovery to therapy. Cancer Discovery, 6:353-367, Apr 2016. URL: https://doi.org/10.1158/2159-8290.cd-15-0894, doi:10.1158/2159-8290.cd-15-0894. This article has 948 citations and is from a highest quality peer-reviewed journal.
12. (sielecki2000cyclindependentkinaseinhibitors pages 5-6): Thais M. Sielecki, John F. Boylan, Pamela A. Benfield, and George L. Trainor. Cyclin-dependent kinase inhibitors: useful targets in cell cycle regulation. Journal of medicinal chemistry, 43 1:1-18, Jan 2000. URL: https://doi.org/10.1021/jm990256j, doi:10.1021/jm990256j. This article has 455 citations and is from a highest quality peer-reviewed journal.
13. (tadesse2018cyclindependentkinase2 pages 4-8): Solomon Tadesse, Elizabeth C. Caldon, Wayne Tilley, and Shudong Wang. Cyclin-dependent kinase 2 inhibitors in cancer therapy: an update. Journal of Medicinal Chemistry, 62:4233-4251, Dec 2018. URL: https://doi.org/10.1021/acs.jmedchem.8b01469, doi:10.1021/acs.jmedchem.8b01469. This article has 250 citations and is from a highest quality peer-reviewed journal.
14. (wood2018structuralinsightsinto pages 1-2): Daniel J. Wood and Jane A. Endicott. Structural insights into the functional diversity of the cdk–cyclin family. Open Biology, Sep 2018. URL: https://doi.org/10.1098/rsob.180112, doi:10.1098/rsob.180112. This article has 266 citations and is from a peer-reviewed journal.
15. (ding2020therolesof pages 1-3): L. Ding, Jiaqi Cao, Wen-Lien Lin, Hongjian Chen, Xianhui Xiong, Hongshun Ao, Min Yu, Jie Lin, and Qing-hua Cui. The roles of cyclin-dependent kinases in cell-cycle progression and therapeutic strategies in human breast cancer. International Journal of Molecular Sciences, 21:1960, Mar 2020. URL: https://doi.org/10.3390/ijms21061960, doi:10.3390/ijms21061960. This article has 615 citations and is from a peer-reviewed journal.
16. (ding2020therolesof pages 5-7): L. Ding, Jiaqi Cao, Wen-Lien Lin, Hongjian Chen, Xianhui Xiong, Hongshun Ao, Min Yu, Jie Lin, and Qing-hua Cui. The roles of cyclin-dependent kinases in cell-cycle progression and therapeutic strategies in human breast cancer. International Journal of Molecular Sciences, 21:1960, Mar 2020. URL: https://doi.org/10.3390/ijms21061960, doi:10.3390/ijms21061960. This article has 615 citations and is from a peer-reviewed journal.
17. (ding2020therolesof pages 7-8): L. Ding, Jiaqi Cao, Wen-Lien Lin, Hongjian Chen, Xianhui Xiong, Hongshun Ao, Min Yu, Jie Lin, and Qing-hua Cui. The roles of cyclin-dependent kinases in cell-cycle progression and therapeutic strategies in human breast cancer. International Journal of Molecular Sciences, 21:1960, Mar 2020. URL: https://doi.org/10.3390/ijms21061960, doi:10.3390/ijms21061960. This article has 615 citations and is from a peer-reviewed journal.
18. (ferrer2006structuralbasisfor pages 3-4): Jean-Luc Ferrer, Jerome Dupuy, Franck Borel, Lilian Jacquamet, Joseph P. Noel, and Vjekoslav Dulic. Structural basis for the modulation of cdk-dependent/independent activity of cyclin d1. Cell Cycle, 5:2760-2768, Nov 2006. URL: https://doi.org/10.4161/cc.5.23.3506, doi:10.4161/cc.5.23.3506. This article has 24 citations and is from a peer-reviewed journal.
19. (ferrer2006structuralbasisfor pages 4-5): Jean-Luc Ferrer, Jerome Dupuy, Franck Borel, Lilian Jacquamet, Joseph P. Noel, and Vjekoslav Dulic. Structural basis for the modulation of cdk-dependent/independent activity of cyclin d1. Cell Cycle, 5:2760-2768, Nov 2006. URL: https://doi.org/10.4161/cc.5.23.3506, doi:10.4161/cc.5.23.3506. This article has 24 citations and is from a peer-reviewed journal.
20. (ferrer2006structuralbasisfor pages 5-6): Jean-Luc Ferrer, Jerome Dupuy, Franck Borel, Lilian Jacquamet, Joseph P. Noel, and Vjekoslav Dulic. Structural basis for the modulation of cdk-dependent/independent activity of cyclin d1. Cell Cycle, 5:2760-2768, Nov 2006. URL: https://doi.org/10.4161/cc.5.23.3506, doi:10.4161/cc.5.23.3506. This article has 24 citations and is from a peer-reviewed journal.
21. (huwe2003smallmoleculesas pages 3-4): Axel Huwe, Ralph Mazitschek, and Athanassios Giannis. Small molecules as inhibitors of cyclin-dependent kinases. Angewandte Chemie, 42 19:2122-38, May 2003. URL: https://doi.org/10.1002/anie.200200540, doi:10.1002/anie.200200540. This article has 235 citations and is from a highest quality peer-reviewed journal.
22. (knockaert2002pharmacologicalinhibitorsof pages 3-4): Marie Knockaert, Paul Greengard, and Laurent Meijer. Pharmacological inhibitors of cyclin-dependent kinases. Trends in Pharmacological Sciences, 23:417-425, Sep 2002. URL: https://doi.org/10.1016/s0165-6147(02)02071-0, doi:10.1016/s0165-6147(02)02071-0. This article has 741 citations and is from a highest quality peer-reviewed journal.
23. (knockaert2002pharmacologicalinhibitorsof pages 4-5): Marie Knockaert, Paul Greengard, and Laurent Meijer. Pharmacological inhibitors of cyclin-dependent kinases. Trends in Pharmacological Sciences, 23:417-425, Sep 2002. URL: https://doi.org/10.1016/s0165-6147(02)02071-0, doi:10.1016/s0165-6147(02)02071-0. This article has 741 citations and is from a highest quality peer-reviewed journal.
24. (lolli2005cak—cyclindependentactivatingkinase pages 6-6): Graziano Lolli and Louise N. Johnson. Cak—cyclin-dependent activating kinase: a key kinase in cell cycle control and a target for drugs? Cell Cycle, 4:565-570, Jan 2005. URL: https://doi.org/10.4161/cc.4.4.1607, doi:10.4161/cc.4.4.1607. This article has 309 citations and is from a peer-reviewed journal.
25. (malumbres2005mammaliancyclindependentkinases pages 1-2): Marcos Malumbres and Mariano Barbacid. Mammalian cyclin-dependent kinases. Trends in Biochemical Sciences, 30:630-641, Nov 2005. URL: https://doi.org/10.1016/j.tibs.2005.09.005, doi:10.1016/j.tibs.2005.09.005. This article has 1760 citations and is from a domain leading peer-reviewed journal.
26. (malumbres2005mammaliancyclindependentkinases pages 3-4): Marcos Malumbres and Mariano Barbacid. Mammalian cyclin-dependent kinases. Trends in Biochemical Sciences, 30:630-641, Nov 2005. URL: https://doi.org/10.1016/j.tibs.2005.09.005, doi:10.1016/j.tibs.2005.09.005. This article has 1760 citations and is from a domain leading peer-reviewed journal.
27. (malumbres2005mammaliancyclindependentkinases pages 6-7): Marcos Malumbres and Mariano Barbacid. Mammalian cyclin-dependent kinases. Trends in Biochemical Sciences, 30:630-641, Nov 2005. URL: https://doi.org/10.1016/j.tibs.2005.09.005, doi:10.1016/j.tibs.2005.09.005. This article has 1760 citations and is from a domain leading peer-reviewed journal.
28. (malumbres2011physiologicalrelevanceof pages 3-4): Marcos Malumbres. Physiological relevance of cell cycle kinases. Physiological Reviews, 91:973-1007, Jul 2011. URL: https://doi.org/10.1152/physrev.00025.2010, doi:10.1152/physrev.00025.2010. This article has 300 citations and is from a highest quality peer-reviewed journal.
29. (malumbres2014cyclindependentkinases pages 1-2): Marcos Malumbres. Cyclin-dependent kinases. Genome Biology, 15:122-122, Jun 2014. URL: https://doi.org/10.1186/gb4184, doi:10.1186/gb4184. This article has 1882 citations and is from a highest quality peer-reviewed journal.
30. (malumbres2014cyclindependentkinases pages 5-6): Marcos Malumbres. Cyclin-dependent kinases. Genome Biology, 15:122-122, Jun 2014. URL: https://doi.org/10.1186/gb4184, doi:10.1186/gb4184. This article has 1882 citations and is from a highest quality peer-reviewed journal.
31. (malumbres2014cyclindependentkinases pages 6-7): Marcos Malumbres. Cyclin-dependent kinases. Genome Biology, 15:122-122, Jun 2014. URL: https://doi.org/10.1186/gb4184, doi:10.1186/gb4184. This article has 1882 citations and is from a highest quality peer-reviewed journal.
32. (malumbres2014cyclindependentkinases pages 8-9): Marcos Malumbres. Cyclin-dependent kinases. Genome Biology, 15:122-122, Jun 2014. URL: https://doi.org/10.1186/gb4184, doi:10.1186/gb4184. This article has 1882 citations and is from a highest quality peer-reviewed journal.
33. (peyressatre2015targetingcyclindependentkinases pages 15-17): Marion Peyressatre, Camille Prével, Morgan Pellerano, and May Morris. Targeting cyclin-dependent kinases in human cancers: from small molecules to peptide inhibitors. Cancers, 7:179-237, Jan 2015. URL: https://doi.org/10.3390/cancers7010179, doi:10.3390/cancers7010179. This article has 402 citations and is from a peer-reviewed journal.
34. (peyressatre2015targetingcyclindependentkinases pages 4-6): Marion Peyressatre, Camille Prével, Morgan Pellerano, and May Morris. Targeting cyclin-dependent kinases in human cancers: from small molecules to peptide inhibitors. Cancers, 7:179-237, Jan 2015. URL: https://doi.org/10.3390/cancers7010179, doi:10.3390/cancers7010179. This article has 402 citations and is from a peer-reviewed journal.
35. (peyressatre2015targetingcyclindependentkinases pages 6-8): Marion Peyressatre, Camille Prével, Morgan Pellerano, and May Morris. Targeting cyclin-dependent kinases in human cancers: from small molecules to peptide inhibitors. Cancers, 7:179-237, Jan 2015. URL: https://doi.org/10.3390/cancers7010179, doi:10.3390/cancers7010179. This article has 402 citations and is from a peer-reviewed journal.
36. (sielecki2000cyclindependentkinaseinhibitors pages 1-2): Thais M. Sielecki, John F. Boylan, Pamela A. Benfield, and George L. Trainor. Cyclin-dependent kinase inhibitors: useful targets in cell cycle regulation. Journal of medicinal chemistry, 43 1:1-18, Jan 2000. URL: https://doi.org/10.1021/jm990256j, doi:10.1021/jm990256j. This article has 455 citations and is from a highest quality peer-reviewed journal.
37. (suryadinata2010controlofcell pages 3-4): Randy Suryadinata, Martin Sadowski, and Boris Sarcevic. Control of cell cycle progression by phosphorylation of cyclin-dependent kinase (cdk) substrates. Bioscience reports, 30 4:243-55, Aug 2010. URL: https://doi.org/10.1042/bsr20090171, doi:10.1042/bsr20090171. This article has 227 citations and is from a peer-reviewed journal.
38. (suryadinata2010controlofcell pages 9-10): Randy Suryadinata, Martin Sadowski, and Boris Sarcevic. Control of cell cycle progression by phosphorylation of cyclin-dependent kinase (cdk) substrates. Bioscience reports, 30 4:243-55, Aug 2010. URL: https://doi.org/10.1042/bsr20090171, doi:10.1042/bsr20090171. This article has 227 citations and is from a peer-reviewed journal.
39. (tadesse2018cyclindependentkinase2 pages 18-23): Solomon Tadesse, Elizabeth C. Caldon, Wayne Tilley, and Shudong Wang. Cyclin-dependent kinase 2 inhibitors in cancer therapy: an update. Journal of Medicinal Chemistry, 62:4233-4251, Dec 2018. URL: https://doi.org/10.1021/acs.jmedchem.8b01469, doi:10.1021/acs.jmedchem.8b01469. This article has 250 citations and is from a highest quality peer-reviewed journal.
40. (wang2017themetabolicfunction pages 1-3): Haizhen Wang, Brandon N. Nicolay, Joel M. Chick, Xueliang Gao, Yan Geng, Hong Ren, Hui Gao, Guizhi Yang, Juliet A. Williams, Jan M. Suski, Mark A. Keibler, Ewa Sicinska, Ulrike Gerdemann, W. Nicholas Haining, Thomas M. Roberts, Kornelia Polyak, Steven P. Gygi, Nicholas J. Dyson, and Piotr Sicinski. The metabolic function of cyclin d3–cdk6 kinase in cancer cell survival. Nature, 546:426-430, Jun 2017. URL: https://doi.org/10.1038/nature22797, doi:10.1038/nature22797. This article has 358 citations and is from a highest quality peer-reviewed journal.
41. (wood2018structuralinsightsinto pages 17-18): Daniel J. Wood and Jane A. Endicott. Structural insights into the functional diversity of the cdk–cyclin family. Open Biology, Sep 2018. URL: https://doi.org/10.1098/rsob.180112, doi:10.1098/rsob.180112. This article has 266 citations and is from a peer-reviewed journal.
42. (wood2018structuralinsightsinto pages 18-19): Daniel J. Wood and Jane A. Endicott. Structural insights into the functional diversity of the cdk–cyclin family. Open Biology, Sep 2018. URL: https://doi.org/10.1098/rsob.180112, doi:10.1098/rsob.180112. This article has 266 citations and is from a peer-reviewed journal.
43. (wood2018structuralinsightsinto pages 2-3): Daniel J. Wood and Jane A. Endicott. Structural insights into the functional diversity of the cdk–cyclin family. Open Biology, Sep 2018. URL: https://doi.org/10.1098/rsob.180112, doi:10.1098/rsob.180112. This article has 266 citations and is from a peer-reviewed journal.
44. (wood2018structuralinsightsinto pages 20-20): Daniel J. Wood and Jane A. Endicott. Structural insights into the functional diversity of the cdk–cyclin family. Open Biology, Sep 2018. URL: https://doi.org/10.1098/rsob.180112, doi:10.1098/rsob.180112. This article has 266 citations and is from a peer-reviewed journal.
45. (wood2018structuralinsightsinto pages 6-7): Daniel J. Wood and Jane A. Endicott. Structural insights into the functional diversity of the cdk–cyclin family. Open Biology, Sep 2018. URL: https://doi.org/10.1098/rsob.180112, doi:10.1098/rsob.180112. This article has 266 citations and is from a peer-reviewed journal.
46. (wood2018structuralinsightsinto pages 7-8): Daniel J. Wood and Jane A. Endicott. Structural insights into the functional diversity of the cdk–cyclin family. Open Biology, Sep 2018. URL: https://doi.org/10.1098/rsob.180112, doi:10.1098/rsob.180112. This article has 266 citations and is from a peer-reviewed journal.
47. (fischer2003cyclindependentkinaseinhibitors pages 1-3): PM Fischer, J Endicott, and L Meijer. Cyclin-dependent kinase inhibitors. Unknown journal, 2003.
48. (knockaert2002pharmacologicalinhibitorsof pages 1-2): Marie Knockaert, Paul Greengard, and Laurent Meijer. Pharmacological inhibitors of cyclin-dependent kinases. Trends in Pharmacological Sciences, 23:417-425, Sep 2002. URL: https://doi.org/10.1016/s0165-6147(02)02071-0, doi:10.1016/s0165-6147(02)02071-0. This article has 741 citations and is from a highest quality peer-reviewed journal.
49. (lolli2005cak—cyclindependentactivatingkinase pages 4-5): Graziano Lolli and Louise N. Johnson. Cak—cyclin-dependent activating kinase: a key kinase in cell cycle control and a target for drugs? Cell Cycle, 4:565-570, Jan 2005. URL: https://doi.org/10.4161/cc.4.4.1607, doi:10.4161/cc.4.4.1607. This article has 309 citations and is from a peer-reviewed journal.
50. (malumbres2005mammaliancyclindependentkinases pages 2-3): Marcos Malumbres and Mariano Barbacid. Mammalian cyclin-dependent kinases. Trends in Biochemical Sciences, 30:630-641, Nov 2005. URL: https://doi.org/10.1016/j.tibs.2005.09.005, doi:10.1016/j.tibs.2005.09.005. This article has 1760 citations and is from a domain leading peer-reviewed journal.