1. Phylogeny  
   Cyclin‐dependent kinase 20 (CDK20), also referred to as cell cycle–related kinase (CCRK), CDK‐activating kinase p42, cell division protein kinase 20, cyclin‐dependent protein kinase H, or cyclin–kinase–activating kinase p42, belongs to the cyclin-dependent kinase family that is classified within the CMGC group of serine/threonine protein kinases, an evolutionarily conserved lineage that includes both classical cell cycle regulators (such as CDK1, CDK2, CDK4, and CDK6) and more atypical members involved in transcriptional and developmental regulation (chowdhury2023cmgckinasesin pages 10-12, alexander2021theconciseguide pages 65-67).  
   Phylogenetic studies indicate that CDK20 has orthologs conserved across a broad range of metazoan species, underscoring its essential roles in regulating cell proliferation and neural development through conserved catalytic and regulatory domains (łukasik2021cyclindependentkinases(cdk) pages 1-2, pluta2024cyclin‐dependentkinasesmasters pages 1-3).  
   Its placement within the CMGC kinome highlights the shared ancestry with other kinases that possess a highly conserved bilobal catalytic domain, with ancestral roots that may extend back to the Last Eukaryotic Common Ancestor (LECA) (chowdhury2023cmgckinasesin pages 4-6, alexanderUnknownyeartheconciseguide pages 4-5).  
   The evolutionary conservation of CDK20’s catalytic core and regulatory motifs reflects its indispensable function in orchestrating both cell cycle transitions and key developmental signaling pathways, including those involved in neural tube patterning (chowdhury2023cmgckinasesin pages 2-4, pluta2024cyclin‐dependentkinasesmasters pages 12-14).  
   Taken together, these findings place CDK20 among an ancient and critical subgroup of CDKs that integrate cell cycle control with specialized functions in ciliary assembly and developmental processes (chowdhury2023cmgckinasesin pages 21-22, alexander2021theconciseguide pages 65-67).
2. Reaction Catalyzed  
   CDK20 catalyzes a phosphorylation reaction characteristic of serine/threonine kinases, where it transfers the γ-phosphate group from ATP to the hydroxyl side-chain of serine or threonine residues on substrate proteins (chowdhury2023cmgckinasesin pages 10-12, tadesse2018cyclindependentkinase2 pages 4-8).  
   Specifically, one of its best-characterized reactions is the phosphorylation of CDK2 at threonine-160 within the activation loop, a post-translational modification that is critical to converting CDK2 from an inactive to an active state, thereby promoting cell cycle progression through the G1/S phase transition (mullerdott2025fromactivityinference pages 48-52, alexander2021theconciseguide pages 65-67).  
   The overall reaction can be represented as: ATP + protein–OH → ADP + protein–O–PO₃²⁻ + H⁺, a reaction fundamental not only to the modulation of cell cycle regulators but also to the maintenance of proper signal transduction events (chowdhury2023cmgckinasesin pages 2-4, tadesse2018cyclindependentkinase2 pages 4-8).  
   In addition to its role in directly activating CDK2, CDK20 is also implicated in modulating the assembly of the primary cilium by phosphorylating or regulating other substrates in complex with regulatory proteins such as TBC1D32, thereby indirectly impacting the activation state of transcription factors like GLI2 in response to Sonic Hedgehog (Shh) signaling (chowdhury2023cmgckinasesin pages 22-24, flax2024illuminationofunderstudied pages 2-3).  
   Thus, the biochemical reaction mediated by CDK20 extends its impact from the control of cell proliferation to the regulation of key developmental signaling cascades through precise phosphorylation events (alexander2021theconciseguide pages 65-67, tadesse2018cyclindependentkinase2 pages 4-8).
3. Cofactor Requirements  
   The kinase activity of CDK20, similar to other members of the CMGC kinase family, is critically dependent on the presence of divalent metal ions for its catalytic function (chowdhury2023cmgckinasesin pages 10-12, łukasik2021cyclindependentkinases(cdk) pages 1-2).  
   Magnesium ions (Mg²⁺) serve as the primary cofactor, playing an essential role in coordinating ATP within the active site and stabilizing the transition state during the phosphoryl transfer reaction (chowdhury2023cmgckinasesin pages 10-12, alexander2021theconciseguide pages 65-67).  
   This requirement for Mg²⁺ not only facilitates the efficient binding and proper orientation of ATP but also neutralizes the negative charges of the phosphate groups, thereby ensuring maximal catalytic proficiency under physiological conditions (chowdhury2023cmgckinasesin pages 10-12, łukasik2021cyclindependentkinases(cdk) pages 1-2).  
   No additional unique cofactors or regulatory molecules, beyond what is typically observed in serine/threonine kinases, have been reported for CDK20, emphasizing the conserved nature of its biochemical mechanism within the CDK family (chowdhury2023cmgckinasesin pages 10-12, alexander2021theconciseguide pages 65-67).
4. Substrate Specificity  
   CDK20 exhibits substrate specificity that is defined primarily by its role in phosphorylating CDK2 at the critical threonine-160 residue, a modification that is essential for the activation of CDK2 and subsequent cell cycle progression (chowdhury2023cmgckinasesin pages 10-12, tadesse2018cyclindependentkinase2 pages 4-8).  
   While the exact consensus motif for CDK20 is not fully characterized, its substrate recognition is consistent with the behavior of other cyclin-dependent kinases, which often prefer serine/threonine residues situated within structural contexts that may include proline-directed motifs or adjacent regulatory sequence elements (pellarin2025cyclindependentproteinkinases pages 2-4, wood2018structuralinsightsinto pages 21-22).  
   In addition to directly phosphorylating CDK2, there is evidence suggesting that CDK20 contributes to the regulation of primary cilium assembly through its interaction with TBC1D32, implying that it may target other substrates involved in the structural organization of the cilium, which in turn affect downstream effectors such as GLI2 in Shh signaling (chowdhury2023cmgckinasesin pages 22-24, flax2024illuminationofunderstudied pages 2-3).  
   Thus, CDK20 likely recognizes substrates that possess structural features analogous to those found in classical CDK targets, with substrate specificity modulated by its interactions with cyclins or cyclin-like partners, even though the precise amino acid motif remains to be comprehensively mapped (pellarin2025cyclindependentproteinkinases pages 2-4, wood2018structuralinsightsinto pages 21-22).
5. Structure  
   The overall structure of CDK20 is dictated by its conserved serine/threonine kinase domain, which adheres to the typical bilobal architecture observed among cyclin-dependent kinases (chowdhury2023cmgckinasesin pages 10-12, wood2018structuralinsightsinto pages 1-2).  
   The N-terminal lobe is generally composed of a series of β-sheets along with an important αC-helix that undergoes conformational rearrangement upon cyclin binding, thereby playing a pivotal role in the activation process (leopold2018optogeneticallycontrolledprotein pages 3-4, alexander2021theconciseguide pages 65-67).  
   In contrast, the larger C-terminal lobe houses critical catalytic motifs including the ATP-binding pocket as well as regions responsible for substrate recognition; within this lobe, conserved motifs such as the DFG (Asp-Phe-Gly) and HRD (His-Arg-Asp) sequences are central to catalysis and are preserved in CDK20 (chowdhury2023cmgckinasesin pages 10-12, pluta2024cyclin‐dependentkinasesmasters pages 12-14).  
   Despite the absence of high-resolution crystallographic data specifically for CDK20, homology models and AlphaFold predictions support a structural organization that is similar to that of other CDKs, with potential additional N- and C-terminal extensions that may mediate interactions with regulatory proteins involved in ciliary assembly, such as TBC1D32 (wood2018structuralinsightsinto pages 1-2, leopold2018optogeneticallycontrolledprotein pages 4-6).  
   These extended regions, while less well conserved than the central kinase domain, are hypothesized to confer unique regulatory properties that link the classical kinase activity with specialized roles in developmental signaling and primary cilium organization (chowdhury2023cmgckinasesin pages 6-8, pluta2024cyclin‐dependentkinasesmasters pages 12-14).
6. Regulation  
   The regulatory mechanisms controlling CDK20 are multifaceted and closely mirror those observed amongst cyclin-dependent kinases (chowdhury2023cmgckinasesin pages 6-8, tadesse2018cyclindependentkinase2 pages 4-8).  
   A central regulatory event is the phosphorylation-dependent activation, which, in the case of CDK20, is exemplified by its ability to phosphorylate CDK2 on threonine-160; this phosphorylation event is essential for inducing the conformational changes that yield an active CDK2 capable of driving cell cycle progression (mullerdott2025fromactivityinference pages 48-52, alexander2021theconciseguide pages 65-67).  
   In addition to this critical activity, CDK20 is regulated through its physical interaction with specific binding partners—most notably TBC1D32—which is believed to facilitate its localization to the primary cilium, thereby integrating its catalytic function with developmental signaling processes (chowdhury2023cmgckinasesin pages 22-24, flax2024illuminationofunderstudied pages 1-2).  
   Moreover, as is typical for CDKs, post-translational modifications such as additional phosphorylation events (possibly at sites outside the catalytic loop) may modulate its kinase activity, subcellular distribution, and interactions with other regulatory proteins, though the complete mapping of these modifications for CDK20 remains an area of active investigation (chowdhury2023cmgckinasesin pages 6-8, janackova2023mechanismusregulacecyklindependentní pages 20-24).  
   Together, these regulation processes underscore the temporal and spatial control of CDK20 activity, ensuring its function is tightly coordinated with both cell cycle progression and ciliary-mediated developmental signaling (flax2024illuminationofunderstudied pages 6-7, ettl2022therenaissanceof pages 1-2).
7. Function  
   CDK20 exhibits a dual functionality that is central to both the control of cell proliferation and the regulation of developmental signaling pathways (chowdhury2023cmgckinasesin pages 10-12, pellarin2025cyclindependentproteinkinases pages 2-4).  
   Its most well-established role is the activation of CDK2 via phosphorylation of threonine-160, a modification critical for the progression of the cell cycle, particularly at the G1/S transition, thereby ensuring proper cell growth and division (tadesse2018cyclindependentkinase2 pages 4-8, alexander2021theconciseguide pages 65-67).  
   In parallel, CDK20 is indispensable for mediating high-level Sonic Hedgehog (Shh) signaling responses during neural development; it achieves this by cooperating with TBC1D32 to regulate the structure of the primary cilium, an organelle that is essential for the activation of GLI2, a transcription factor pivotal to the orchestration of neural tube patterning (chowdhury2023cmgckinasesin pages 22-24, flax2024illuminationofunderstudied pages 2-3).  
   By coordinating the assembly of the ciliary membrane and axoneme, CDK20 effectively integrates mitogenic signals with developmental cues, linking the activation status of CDK2 to broader cellular programs that govern tissue differentiation and organogenesis (mullerdott2025fromactivityinference pages 48-52, pellarin2025cyclindependentproteinkinases pages 18-19).  
   Furthermore, aberrant expression of CDK20 has been associated with oncogenic processes in various tumor types, indicating that its dysregulated activity may not only promote uncontrolled cell proliferation but also contribute to developmental abnormalities and cancer progression (pellarin2025cyclindependentproteinkinases pages 54-55, southekal2021integrativeanalysisof pages 19-25).  
   Thus, CDK20 functions as a key signaling hub that connects the regulation of the cell cycle with intricate developmental pathways, making it a subject of considerable interest in both basic biology and translational research aimed at cancer therapeutics (chowdhury2023cmgckinasesin pages 10-12, pluta2024cyclin‐dependentkinasesmasters pages 12-14).
8. Other Comments  
   Despite the significant roles played by CDK20 in the regulation of cell division and developmental signaling, it remains relatively underexplored compared to more thoroughly characterized CDKs, and there is currently a scarcity of specific inhibitors that directly target this kinase (flax2024illuminationofunderstudied pages 6-7, ettl2022therenaissanceof pages 1-2).  
   Recent studies have begun to emphasize CDK20’s potential as an oncogenic driver in various cancers, with overexpression correlating with increased cell proliferation and poor clinical outcomes, thereby positioning it as a promising candidate for therapeutic intervention in oncological settings (pellarin2025cyclindependentproteinkinases pages 56-56, southekal2021integrativeanalysisof pages 19-25).  
   The interplay between CDK20 and developmental signals, particularly through its modulation of primary cilium structure and Shh signaling, may also have important implications in understanding congenital neurological disorders and other developmental maladies (chowdhury2023cmgckinasesin pages 22-24, leopold2018optogeneticallycontrolledprotein pages 4-6).  
   Furthermore, advanced computational approaches such as AlphaFold-based structural predictions are now being harnessed to gain deeper insights into the structural dynamics of CDK20, which could eventually facilitate the design of novel, highly selective inhibitors (wood2018structuralinsightsinto pages 21-22, karimbayli2024insightsintothe pages 15-17).  
   Ongoing research efforts are focused on mapping the full spectrum of post-translational modifications on CDK20 and characterizing its interactions with potential cyclin partners, with the aim of elucidating the detailed molecular mechanisms underlying its regulation and function in both normal and pathological contexts (chowdhury2023cmgckinasesin pages 6-8, janackova2023mechanismusregulacecyklindependentní pages 20-24).
9. References
10. chowdhury2023cmgckinasesin pages 10-12
11. chowdhury2023cmgckinasesin pages 22-24
12. chowdhury2023cmgckinasesin pages 4-6
13. chowdhury2023cmgckinasesin pages 6-8
14. flax2024illuminationofunderstudied pages 1-2
15. flax2024illuminationofunderstudied pages 2-3
16. leopold2018optogeneticallycontrolledprotein pages 3-4
17. alexander2021theconciseguide pages 65-67
18. pluta2024cyclin‐dependentkinasesmasters pages 12-14
19. łukasik2021cyclindependentkinases(cdk) pages 1-2
20. tadesse2018cyclindependentkinase2 pages 4-8
21. mullerdott2025fromactivityinference pages 48-52
22. pellarin2025cyclindependentproteinkinases pages 2-4
23. pellarin2025cyclindependentproteinkinases pages 18-19
24. pellarin2025cyclindependentproteinkinases pages 54-55
25. pellarin2025cyclindependentproteinkinases pages 56-56
26. southekal2021integrativeanalysisof pages 19-25
27. wood2018structuralinsightsinto pages 1-2
28. wood2018structuralinsightsinto pages 21-22
29. karimbayli2024insightsintothe pages 15-17
30. alexanderUnknownyeartheconciseguide pages 4-5
31. janackova2023mechanismusregulacecyklindependentní pages 20-24
32. flax2024illuminationofunderstudied pages 6-7
33. ettl2022therenaissanceof pages 1-2
34. southekal2021integrativeanalysisof pages 19-25

References

1. (chowdhury2023cmgckinasesin pages 10-12): Iftekhar Chowdhury, Giovanna Dashi, and Salla Keskitalo. Cmgc kinases in health and cancer. Cancers, 15:3838, Jul 2023. URL: https://doi.org/10.3390/cancers15153838, doi:10.3390/cancers15153838. This article has 18 citations and is from a peer-reviewed journal.
2. (chowdhury2023cmgckinasesin pages 22-24): Iftekhar Chowdhury, Giovanna Dashi, and Salla Keskitalo. Cmgc kinases in health and cancer. Cancers, 15:3838, Jul 2023. URL: https://doi.org/10.3390/cancers15153838, doi:10.3390/cancers15153838. This article has 18 citations and is from a peer-reviewed journal.
3. (chowdhury2023cmgckinasesin pages 4-6): Iftekhar Chowdhury, Giovanna Dashi, and Salla Keskitalo. Cmgc kinases in health and cancer. Cancers, 15:3838, Jul 2023. URL: https://doi.org/10.3390/cancers15153838, doi:10.3390/cancers15153838. This article has 18 citations and is from a peer-reviewed journal.
4. (chowdhury2023cmgckinasesin pages 6-8): Iftekhar Chowdhury, Giovanna Dashi, and Salla Keskitalo. Cmgc kinases in health and cancer. Cancers, 15:3838, Jul 2023. URL: https://doi.org/10.3390/cancers15153838, doi:10.3390/cancers15153838. This article has 18 citations and is from a peer-reviewed journal.
5. (flax2024illuminationofunderstudied pages 1-2): Raymond G. Flax, Peter Rosston, Cecilia Rocha, Brian Anderson, Jacob L. Capener, Thomas M. Durcan, David H. Drewry, Panagiotis Prinos, and Alison D. Axtman. Illumination of understudied ciliary kinases. Frontiers in Molecular Biosciences, Mar 2024. URL: https://doi.org/10.3389/fmolb.2024.1352781, doi:10.3389/fmolb.2024.1352781. This article has 5 citations and is from a peer-reviewed journal.
6. (flax2024illuminationofunderstudied pages 2-3): Raymond G. Flax, Peter Rosston, Cecilia Rocha, Brian Anderson, Jacob L. Capener, Thomas M. Durcan, David H. Drewry, Panagiotis Prinos, and Alison D. Axtman. Illumination of understudied ciliary kinases. Frontiers in Molecular Biosciences, Mar 2024. URL: https://doi.org/10.3389/fmolb.2024.1352781, doi:10.3389/fmolb.2024.1352781. This article has 5 citations and is from a peer-reviewed journal.
7. (flax2024illuminationofunderstudied pages 6-7): Raymond G. Flax, Peter Rosston, Cecilia Rocha, Brian Anderson, Jacob L. Capener, Thomas M. Durcan, David H. Drewry, Panagiotis Prinos, and Alison D. Axtman. Illumination of understudied ciliary kinases. Frontiers in Molecular Biosciences, Mar 2024. URL: https://doi.org/10.3389/fmolb.2024.1352781, doi:10.3389/fmolb.2024.1352781. This article has 5 citations and is from a peer-reviewed journal.
8. (janackova2023mechanismusregulacecyklindependentní pages 20-24): Z Janáčková. Mechanismus regulace cyklin-dependentní kinasy 16 prostřednictvím komplexu cyklin y/14-3-3. Unknown journal, 2023.
9. (mullerdott2025fromactivityinference pages 48-52): S Müller-Dott. From activity inference to multi-omics network contextualization: deciphering cellular signaling and disease mechanisms. Unknown journal, 2025.
10. (pluta2024cyclin‐dependentkinasesmasters pages 12-14): Aleksandra J. Pluta, Cécilia Studniarek, Shona Murphy, and Chris J. Norbury. Cyclin‐dependent kinases: masters of the eukaryotic universe. WIREs RNA, Sep 2024. URL: https://doi.org/10.1002/wrna.1816, doi:10.1002/wrna.1816. This article has 19 citations.
11. (łukasik2021cyclindependentkinases(cdk) pages 1-2): Paweł Łukasik, Michał Załuski, and Izabela Gutowska. Cyclin-dependent kinases (cdk) and their role in diseases development–review. International Journal of Molecular Sciences, 22:2935, Mar 2021. URL: https://doi.org/10.3390/ijms22062935, doi:10.3390/ijms22062935. This article has 198 citations and is from a peer-reviewed journal.
12. (alexander2021theconciseguide pages 65-67): Stephen P H Alexander, Doriano Fabbro, Eamonn Kelly, Alistair Mathie, John A Peters, Emma L Veale, Jane F Armstrong, Elena Faccenda, Simon D Harding, Adam J Pawson, Christopher Southan, Jamie A Davies, Stephanie Annett, Detlev Boison, Kathryn Elisa Burns, Carmen Dessauer, Jürg Gertsch, Nuala Ann Helsby, Angelo A. Izzo, Doris Koesling, Rennolds Ostrom, Andreas Papapetropoulos, Nigel J. Pyne, Susan Pyne, Tracy Robson, Michael Russwurm, Roland Seifert, Johannes‐Peter Stasch, Csaba Szabo, Mario van der Stelt, Albert van der Vliet, Val Watts, and Szu Shen Wong. The concise guide to pharmacology 2021/22: enzymes. British Journal of Pharmacology, Oct 2021. URL: https://doi.org/10.1111/bph.15542, doi:10.1111/bph.15542. This article has 385 citations and is from a highest quality peer-reviewed journal.
13. (alexanderUnknownyeartheconciseguide pages 4-5): SPH Alexander. The concise guide to pharmacology 2019/20. Unknown journal, Unknown year.
14. (chowdhury2023cmgckinasesin pages 2-4): Iftekhar Chowdhury, Giovanna Dashi, and Salla Keskitalo. Cmgc kinases in health and cancer. Cancers, 15:3838, Jul 2023. URL: https://doi.org/10.3390/cancers15153838, doi:10.3390/cancers15153838. This article has 18 citations and is from a peer-reviewed journal.
15. (chowdhury2023cmgckinasesin pages 21-22): Iftekhar Chowdhury, Giovanna Dashi, and Salla Keskitalo. Cmgc kinases in health and cancer. Cancers, 15:3838, Jul 2023. URL: https://doi.org/10.3390/cancers15153838, doi:10.3390/cancers15153838. This article has 18 citations and is from a peer-reviewed journal.
16. (ettl2022therenaissanceof pages 1-2): Tobias Ettl, Daniela Schulz, and Richard Bauer. The renaissance of cyclin dependent kinase inhibitors. Cancers, 14:293, Jan 2022. URL: https://doi.org/10.3390/cancers14020293, doi:10.3390/cancers14020293. This article has 58 citations and is from a peer-reviewed journal.
17. (karimbayli2024insightsintothe pages 15-17): Javad Karimbayli, Ilenia Pellarin, Barbara Belletti, and Gustavo Baldassarre. Insights into the structural and functional activities of forgotten kinases: pctaires cdks. Molecular Cancer, Jun 2024. URL: https://doi.org/10.1186/s12943-024-02043-6, doi:10.1186/s12943-024-02043-6. This article has 4 citations and is from a highest quality peer-reviewed journal.
18. (leopold2018optogeneticallycontrolledprotein pages 3-4): Anna V. Leopold, Konstantin G. Chernov, and Vladislav V. Verkhusha. Optogenetically controlled protein kinases for regulation of cellular signaling. Chemical Society reviews, 47 7:2454-2484, Apr 2018. URL: https://doi.org/10.1039/c7cs00404d, doi:10.1039/c7cs00404d. This article has 79 citations and is from a highest quality peer-reviewed journal.
19. (leopold2018optogeneticallycontrolledprotein pages 4-6): Anna V. Leopold, Konstantin G. Chernov, and Vladislav V. Verkhusha. Optogenetically controlled protein kinases for regulation of cellular signaling. Chemical Society reviews, 47 7:2454-2484, Apr 2018. URL: https://doi.org/10.1039/c7cs00404d, doi:10.1039/c7cs00404d. This article has 79 citations and is from a highest quality peer-reviewed journal.
20. (pellarin2025cyclindependentproteinkinases pages 18-19): Ilenia Pellarin, Alessandra Dall’Acqua, Andrea Favero, Ilenia Segatto, Valentina Rossi, Nicole Crestan, Javad Karimbayli, Barbara Belletti, and Gustavo Baldassarre. Cyclin-dependent protein kinases and cell cycle regulation in biology and disease. Signal Transduction and Targeted Therapy, Jan 2025. URL: https://doi.org/10.1038/s41392-024-02080-z, doi:10.1038/s41392-024-02080-z. This article has 20 citations and is from a peer-reviewed journal.
21. (pellarin2025cyclindependentproteinkinases pages 2-4): Ilenia Pellarin, Alessandra Dall’Acqua, Andrea Favero, Ilenia Segatto, Valentina Rossi, Nicole Crestan, Javad Karimbayli, Barbara Belletti, and Gustavo Baldassarre. Cyclin-dependent protein kinases and cell cycle regulation in biology and disease. Signal Transduction and Targeted Therapy, Jan 2025. URL: https://doi.org/10.1038/s41392-024-02080-z, doi:10.1038/s41392-024-02080-z. This article has 20 citations and is from a peer-reviewed journal.
22. (pellarin2025cyclindependentproteinkinases pages 54-55): Ilenia Pellarin, Alessandra Dall’Acqua, Andrea Favero, Ilenia Segatto, Valentina Rossi, Nicole Crestan, Javad Karimbayli, Barbara Belletti, and Gustavo Baldassarre. Cyclin-dependent protein kinases and cell cycle regulation in biology and disease. Signal Transduction and Targeted Therapy, Jan 2025. URL: https://doi.org/10.1038/s41392-024-02080-z, doi:10.1038/s41392-024-02080-z. This article has 20 citations and is from a peer-reviewed journal.
23. (pellarin2025cyclindependentproteinkinases pages 56-56): Ilenia Pellarin, Alessandra Dall’Acqua, Andrea Favero, Ilenia Segatto, Valentina Rossi, Nicole Crestan, Javad Karimbayli, Barbara Belletti, and Gustavo Baldassarre. Cyclin-dependent protein kinases and cell cycle regulation in biology and disease. Signal Transduction and Targeted Therapy, Jan 2025. URL: https://doi.org/10.1038/s41392-024-02080-z, doi:10.1038/s41392-024-02080-z. This article has 20 citations and is from a peer-reviewed journal.
24. (pluta2024cyclin‐dependentkinasesmasters pages 1-3): Aleksandra J. Pluta, Cécilia Studniarek, Shona Murphy, and Chris J. Norbury. Cyclin‐dependent kinases: masters of the eukaryotic universe. WIREs RNA, Sep 2024. URL: https://doi.org/10.1002/wrna.1816, doi:10.1002/wrna.1816. This article has 19 citations.
25. (southekal2021integrativeanalysisof pages 19-25): S Southekal. Integrative analysis of multi-omics kinome data and virtual screening of identified targets with pan-cancer application. Unknown journal, 2021.
26. (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 247 citations and is from a highest quality peer-reviewed journal.
27. (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 264 citations and is from a peer-reviewed journal.