## 1. Phylogeny

Serine/threonine‐protein kinase DCLK1, encoded by the DCLK1 gene and alternatively known as DCAMKL1, DCDC3A, or KIAA0369, represents an intriguing evolutionary fusion of functions that combine characteristics of microtubule‐binding proteins with those of serine/threonine kinases (carli2022thefunctionof pages 157-160). DCLK1 traces its evolutionary lineage to a distinct subgroup of kinases that share a common ancestry with the calcium/calmodulin‐dependent kinase (CAMK) family, yet it diverges from classical CAMKs by incorporating one or more N‐terminal doublecortin (DCX) domains that are specialized for microtubule binding (carli2023structureguidedpredictionof pages 1-2). The presence of these DCX domains in DCLK1 is particularly noteworthy because they mirror the structural features found in the doublecortin (DCX) protein, which plays a crucial role in neuronal migration during embryonic development; this suggests that DCLK1 has maintained an ancestral function related to the regulation of microtubule dynamics across higher eukaryotes (carli2022thefunctionofa pages 160-180, carli2023structureguidedpredictionof pages 16-17). Furthermore, orthologs of DCLK1 have been identified in a wide range of mammalian species, underscoring the conservation of both its kinase domain and DCX domains throughout vertebrate evolution (carli2022thefunctionof pages 210-213, carli2023structureguidedpredictionof pages 1-2). The evolutionary diversification of DCLK1 is also evident in the existence of multiple isoforms, which include full-length variants (such as DCLK1-740 and DCLK1-729) that contain both the microtubule-binding and catalytic domains, as well as shorter kinase-only isoforms (for example, DCLK1-433 and DCLK1-422) that result from alternative promoter usage and differential splicing (carli2022thefunctionof pages 160-180, carli2022thefunctionofa pages 210-213). These isoforms illustrate how alternative splicing and promoter selection have been conserved as mechanisms to fine-tune DCLK1’s function in a tissue-specific manner, thereby expanding its roles from neurodevelopment into other cellular contexts such as cancer biology (carli2022thefunctionof pages 160-180, carli2023structureguidedpredictionof pages 16-17). In summary, DCLK1 occupies a unique evolutionary niche by merging a catalytic kinase domain with microtubule-regulatory DCX domains, a combination that has been maintained across species, reflecting its essential role in modulating neuronal migration, microtubule dynamics, and cell signaling (carli2022thefunctionof pages 157-160, carli2023structureguidedpredictionof pages 1-2).

## 2. Reaction Catalyzed

DCLK1 catalyzes a phosphorylation reaction characteristic of serine/threonine protein kinases, with the overall chemical equation described as:  
  ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (ferguson2020discoveryofa pages 1-2).  
In this reaction, DCLK1 transfers the γ-phosphate group from ATP to the hydroxyl group of serine or threonine residues on its substrates, thereby altering the substrates’ conformation, activity, and interaction potential (ferguson2020discoveryofa pages 1-2). The phosphorylation event serves as a post-translational modification that is central to the regulation of various intracellular signaling pathways, and it plays a critical role in modulating microtubule dynamics and cellular motility (carli2023structureguidedpredictionof pages 8-9). Experimental studies, particularly in cancer cell models, have provided evidence that DCLK1-mediated phosphorylation regulates the activity of key proteins involved in microtubule stabilization, such as MAP7D1, whose phosphorylation state directly influences microtubule assembly and dynamics (liu2020chemicalbiologytoolkit pages 10-11, carli2023structureguidedpredictionof pages 8-9). In addition to phosphorylating exogenous substrates, DCLK1 is also capable of autophosphorylation; that is, it can phosphorylate specific serine/threonine residues within its own structure, especially within its regulatory C-terminal tail, thereby modulating its own enzymatic activity and subcellular localization (rogers2020autoregulatorycontrolof pages 1-3). This autophosphorylation is integral to the feedback regulation of DCLK1: by modifying its own phosphorylation state, DCLK1 adjusts the balance between its active and inactive conformations, which in turn regulates its interaction with microtubules and other signaling proteins (rogers2020autoregulatorycontrolof pages 1-3). The mechanistic steps of DCLK1’s catalytic reaction involve the binding of ATP at the kinase active site, precise alignment of the catalytic residues to stabilize the transition state, and subsequent release of ADP following the transfer of the phosphate group to the substrate’s serine or threonine hydroxyl group (carli2023structureguidedpredictionof pages 8-9, ferguson2020discoveryofa pages 1-2). Overall, the phosphorylation reaction catalyzed by DCLK1 constitutes the molecular basis for its role in regulating key cellular processes such as microtubule remodeling, neuronal migration, and in the aberrant signaling observed in oncogenesis (carli2022thefunctionof pages 160-180).

## 3. Cofactor Requirements

The catalytic activity of DCLK1 is dependent on the presence of specific divalent metal ions that serve as essential cofactors, with magnesium (Mg²⁺) being the primary metal ion required for its function (ferguson2020discoveryofa pages 1-2). Mg²⁺ is critical as it helps neutralize the negative charge on ATP, thus facilitating its proper binding and positioning within the kinase active site for efficient phosphoryl transfer to targeted substrates (ferguson2020discoveryofa pages 1-2). In certain experimental kinase assays, manganese (Mn²⁺) has been used as an alternative cofactor; however, empirical evidence strongly supports the physiological relevance of Mg²⁺ in mediating DCLK1’s catalysis (luo2023doublecortinlikekinase1 pages 13-14). The coordination of Mg²⁺ with ATP is not only vital for catalysis but also contributes to the stabilization of the nucleotide-binding loop (or P-loop) within the kinase domain, ensuring that key catalytic residues are properly oriented to engage in the phosphoryl transfer reaction (carli2023structureguidedpredictionof pages 8-9, venkat2023mechanisticandevolutionary pages 16-17). This metal-ion dependency is consistent with the broader biochemical mechanism of serine/threonine kinases, underscoring the conserved role of Mg²⁺ as a catalytic cofactor across the kinase superfamily (ferguson2020discoveryofa pages 1-2). As a result, any modulation of intracellular Mg²⁺ levels or disruption of its coordination with the ATP-binding site could have a direct impact on the enzymatic efficiency of DCLK1, thereby influencing its downstream signaling functions (luo2023doublecortinlikekinase1 pages 13-14). In sum, the cofactor requirements for DCLK1 underscore the centrality of Mg²⁺ in its catalytic process, ensuring that the phosphorylation of substrates occurs with the high efficiency necessary for its diverse cellular roles (carli2023structureguidedpredictionof pages 8-9, ferguson2020discoveryofa pages 1-2).

## 4. Substrate Specificity

DCLK1 exhibits substrate specificity typical of serine/threonine kinases, targeting serine and threonine residues within a variety of protein substrates that are central to cytoskeletal organization and intracellular signaling (carli2022thefunctionof pages 160-180, carli2022thefunctionofa pages 160-180). Phosphoproteomic studies performed in cancer cell models have revealed that modulation of DCLK1 activity leads to significant changes in the phosphorylation status of proteins such as MAP7D1, a microtubule-associated protein whose phosphorylation is critical for microtubule nucleation and stabilization (carli2023structureguidedpredictionof pages 8-9, liu2020chemicalbiologytoolkit pages 10-11). Although a definitive consensus sequence has not been fully established, the substrate preferences of DCLK1 appear to involve motifs resembling those recognized by other members of the CAMK family; these motifs typically include basic amino acid residues positioned near the target serine or threonine residue (liu2020chemicalbiologytoolkit pages 10-11, carli2023structureguidedpredictionof pages 8-9). In addition, DCLK1 is known to undergo autophosphorylation, wherein it phosphorylates residues within its own regulatory regions—most notably within the C-terminal tail—thus providing a self-regulatory mechanism that impacts its overall activity and microtubule-binding properties (rogers2020autoregulatorycontrolof pages 1-3). The dual capability to phosphorylate both exogenous substrates and itself allows DCLK1 to directly control not only the activity of downstream proteins involved in microtubule dynamics and cellular transport, but also to fine-tune its own catalytic state for optimal function (rogers2020autoregulatorycontrolof pages 1-3, carli2023structureguidedpredictionof pages 16-17). Overall, while the complete spectrum of physiological substrates for DCLK1 is still being elucidated, current evidence consistently points to a preference for substrates that regulate cytoskeletal remodeling, vesicle trafficking, and signal transduction pathways related to neuronal migration and oncogenic transformation (carli2022thefunctionof pages 160-180, carli2023structureguidedpredictionof pages 8-9).

## 5. Structure

DCLK1 is architecturally sophisticated, with a modular domain organization that equips it for its dual roles in microtubule regulation and catalytic signaling. The full-length isoforms—commonly referred to as DCLK1-740 or DCLK1-729—comprise several distinct structural elements. At the N-terminus, two tandem doublecortin (DCX) domains, denoted as DC1 and DC2, are present; these domains are responsible for binding to microtubules and influencing their polymerization and stability (carli2022thefunctionof pages 38-42, carli2023structureguidedpredictionof pages 4-6). The DC1 domain generally shows a preference for associating with polymerized tubulin, while the DC2 domain is more versatile, binding both polymerized and soluble tubulin, which suggests a role in microtubule nucleation and dynamic stabilization (carli2022thefunctionof pages 38-42). Interposed between these DCX domains and the catalytic kinase domain is a highly phosphorylated PEST sequence region that is rich in proline, glutamic acid, serine, and threonine residues; this flexible linker not only confers structural adaptability but also serves as a regulatory hub for numerous phosphorylation events that influence both localization and protein–protein interactions (carli2022thefunctionof pages 38-42, carli2023structureguidedpredictionof pages 8-9).  
The central catalytic domain of DCLK1, spanning roughly residues 382–648 in many isoforms, adopts the classic bilobal structure characteristic of serine/threonine kinases. Its N-terminal lobe is predominantly composed of β-strands while the C-terminal lobe is mainly α-helical, together forming the ATP-binding cleft where catalysis occurs (patel2016biochemicalandstructural pages 1-3, patel2016biochemicalandstructural pages 3-4). Within this domain, several conserved catalytic residues are essential for activity, including a lysine that participates in ATP coordination and forms a salt bridge with a glutamate to maintain the active-like “DFG-in” conformation observed in crystal structures (patel2016biochemicalandstructural pages 1-3, carli2023structureguidedpredictionof pages 8-9).  
Downstream of the kinase domain, DCLK1 contains a C-terminal regulatory tail that exerts autoinhibitory control over its catalytic activity. Structural studies and crystallographic data have demonstrated that this tail can engage intramolecularly with the kinase domain, thereby occluding the ATP-binding site and modulating substrate access. A key residue within this region—threonine 688—is subject to autophosphorylation, and when phosphorylated, it helps to dampen the kinase’s activity while also protecting the adjacent DCX domains from hyperphosphorylation that could compromise microtubule binding (rogers2020autoregulatorycontrolof pages 1-3, carli2023structureguidedpredictionof pages 8-9). High-resolution X-ray crystallography of the isolated kinase domain has revealed an active conformation stabilized by coordination of a sulfate group that mimics the phosphorylated state, providing further insight into the molecular mechanics of DCLK1’s enzymatic function (patel2016biochemicalandstructural pages 1-3, patel2016biochemicalandstructural pages 3-4). In addition, recent computational models from AlphaFold have depicted how intrinsically disordered regions, such as the PEST sequence and segments of the C-terminal tail, might dynamically interact with the structured domains to fine-tune activity and subcellular localization (carli2023structureguidedpredictionof pages 1-2, venkat2023mechanisticandevolutionary pages 24-25). Overall, the structural organization of DCLK1, integrating its DCX domains, the PEST linker, the catalytic kinase core, and the regulatory C-terminal tail, provides a versatile framework that permits both microtubule modulation and the phosphorylation of diverse substrates in response to cellular cues (carli2023structureguidedpredictionof pages 4-6, patel2016biochemicalandstructural pages 3-4).

## 6. Regulation

The regulation of DCLK1 is achieved through an intricate interplay of intramolecular interactions and post-translational modifications that modulate both its catalytic activity and its microtubule-binding properties. Central to this regulatory network is the autoinhibitory role of the C-terminal tail, which interacts with the kinase domain to limit access to the ATP-binding site and maintain DCLK1 in a relatively low-activity state in the absence of activating signals (rogers2020autoregulatorycontrolof pages 1-3, rogers2021autoregulatorycontrolof pages 1-2). Autophosphorylation plays a pivotal role in the self-regulation of DCLK1; the enzyme can phosphorylate specific residues within its own structure, most notably threonine 688 (T688) in the C-terminal tail, thereby functioning as a molecular switch that modulates its enzymatic activity (rogers2020autoregulatorycontrolof pages 1-3, carli2023structureguidedpredictionof pages 8-9). Once T688 is phosphorylated, it acts as a negative regulator that prevents excessive phosphorylation of the DCX domains, particularly within the DC2 region, which is critical for maintaining microtubule binding affinity (rogers2020autoregulatorycontrolof pages 1-3, carli2023structureguidedpredictionof pages 8-9). Experimental mutagenesis studies have demonstrated that deletion of the C-terminal tail or mutation of T688 results in a hyperphosphorylated state that compromises microtubule association, underscoring the importance of this autoinhibitory mechanism (rogers2020autoregulatorycontrolof pages 1-3).  
Beyond its autophosphorylation, DCLK1 is also regulated by extrinsic kinases such as CDK5, GSK3β, ERK, JNK, and PKA. These kinases have been implicated in phosphorylating DCLK1 at various sites, thereby altering its subcellular localization, catalytic activity, and interaction with the cytoskeleton (carli2022thefunctionof pages 38-42, venkat2023mechanisticandevolutionary pages 18-19). For instance, phosphorylation by these external kinases may promote conformational changes that facilitate the release of the autoinhibitory tail and enhance catalytic output in response to cellular signals such as calcium fluxes. Additionally, proteolytic processing—mediated by calcium-sensitive proteases like calpain—has been observed, generating distinct DCLK1 fragments that may exhibit altered functions or regulatory properties, particularly during neuronal development (carli2022thefunctionof pages 38-42, rogers2021autoregulatorycontrolof pages 20-21). Overall, the regulation of DCLK1 is multifaceted and finely tuned by both intramolecular autoinhibitory mechanisms and extrinsic phosphorylation events, enabling the kinase to rapidly adjust its activity in accordance with cellular needs in processes as diverse as neuronal migration and oncogenic transformation (carli2023structureguidedpredictionof pages 8-9, rogers2020autoregulatorycontrolof pages 1-3, venkat2023mechanisticandevolutionary pages 22-23).

## 7. Function

DCLK1 exerts a broad range of biological functions that are fundamentally linked to its capabilities as both a microtubule-associated protein and a serine/threonine kinase. In the context of neurodevelopment, DCLK1 plays an essential role in neuronal migration and cortical formation. Its DCX domains bind to microtubules, facilitating their nucleation and stabilization, which is critical for directing the migration of neurons during brain development as well as for establishing proper dendritic architecture (carli2023structureguidedpredictionof pages 16-17, luo2023doublecortinlikekinase1 pages 13-14). Disruption of DCLK1 function in developing neurons—through knockdown or genetic mutation—results in mispositioned cells, aberrant axonal outgrowth, and defective dendritic formation, thereby underscoring its vital contribution to the establishment of neural networks (carli2022thefunctionof pages 157-160, carli2022thefunctionof pages 38-42).  
In addition to its neurodevelopmental roles, DCLK1 has emerged as a critical player in oncogenesis. In various cancers, including pancreatic, gastric, and colorectal carcinomas, DCLK1 is often overexpressed and is utilized as a marker for cancer stem cells, which are thought to drive tumor initiation, metastasis, and resistance to therapy (carli2022thefunctionofa pages 210-213, carli2022thefunctionof pages 160-180). The kinase activity of DCLK1 in these contexts influences several signaling pathways that govern epithelial-to-mesenchymal transition (EMT), cytoskeletal reorganization, and vesicular trafficking, thereby enhancing the invasive and proliferative properties of tumor cells (ferguson2020discoveryofa pages 1-2, carli2023structureguidedpredictionof pages 16-17). Moreover, in the mature nervous system, DCLK1 is implicated in processes such as synaptic plasticity and intracellular transport, with its regulation of microtubule dynamics contributing to the maintenance of synaptic organization and neuronal survival under stress conditions (carli2022thefunctionof pages 248-250, carli2022thefunctionof pages 38-42).  
Outside the nervous system, DCLK1 is expressed in specialized cell types such as tuft cells in the gastrointestinal tract, where it is associated with stem cell functions and tissue repair. In this context, aberrant expression of DCLK1 can promote oncogenic transformation by supporting the self-renewal and proliferative capacities of cancer stem cells (carli2022thefunctionof pages 248-250, carli2023structureguidedpredictionof pages 16-17). Thus, through its multifaceted roles—in regulating microtubule dynamics, orchestrating phosphorylation-dependent signal transduction, and modulating both neurodevelopmental and oncogenic processes—DCLK1 serves as a central hub in cellular homeostasis and represents a crucial target for therapeutic intervention in diseases ranging from neurodevelopmental disorders to aggressive cancers (carli2022thefunctionofa pages 210-213, ferguson2020discoveryofa pages 1-2).

## 8. Other Comments

Emerging studies continue to highlight the potential of DCLK1 as a therapeutic target, particularly in the field of oncology. Small molecule inhibitors such as DCLK1‑IN‑1 and XMD8‑92 have shown promising preclinical efficacy by selectively inhibiting DCLK1’s kinase activity, which in turn has been linked to reduced epithelial-to-mesenchymal transition (EMT) and diminished cancer stem cell properties in various tumor models (ferguson2020discoveryofa pages 1-2, carli2022thefunctionof pages 248-250). The therapeutic interest in these inhibitors is heightened by the observation that DCLK1 expression and activity are frequently elevated in tumors, where they contribute to aggressive phenotypes and poor clinical outcomes.  
Furthermore, the existence of multiple DCLK1 isoforms—ranging from full-length variants that include both the DCX domains and the kinase domain, to shorter isoforms lacking the microtubule-binding domains—adds a layer of functional complexity that may influence the efficacy of targeted therapies (carli2022thefunctionof pages 160-180, carli2022thefunctionofa pages 160-180). Isoform-specific expression patterns have been noted in different tissue contexts, suggesting that tailored therapeutic strategies could be developed to selectively target the oncogenic forms of DCLK1 in cancer while sparing its physiological functions in the nervous system and other tissues (carli2023structureguidedpredictionof pages 8-9, rogers2020autoregulatorycontrolof pages 1-3).  
Moreover, recent advancements in high-resolution structural studies and chemical biology toolkits are paving the way for a more thorough elucidation of DCLK1’s substrate repertoire and downstream signaling networks, which will further inform drug design efforts. The integration of advanced proteomic techniques with structural approaches holds promise for identifying novel substrates and interaction partners, thereby expanding our understanding of how DCLK1 dysregulation drives pathological processes (liu2020chemicalbiologytoolkit pages 10-11, venkat2023mechanisticandevolutionary pages 23-24). These research endeavors, coupled with efforts to decipher the mechanisms underlying the autoregulatory control of DCLK1, underscore its multifaceted nature and reinforce its status as a critical node in the regulation of cellular dynamics and tumor progression (rogers2021autoregulatorycontrolof pages 20-21, venkat2023mechanisticandevolutionary pages 24-25).

## 9. References

1. carli2022thefunctionof pages 157-160
2. carli2022thefunctionof pages 160-180
3. carli2022thefunctionof pages 210-213
4. carli2022thefunctionof pages 248-250
5. carli2022thefunctionof pages 38-42
6. carli2022thefunctionofa pages 160-180
7. carli2022thefunctionofa pages 210-213
8. carli2023structureguidedpredictionof pages 1-2
9. carli2023structureguidedpredictionof pages 2-4
10. carli2023structureguidedpredictionof pages 4-6
11. carli2023structureguidedpredictionof pages 8-9
12. carli2023structureguidedpredictionof pages 16-17
13. carli2023structureguidedpredictionof pages 19-20
14. ferguson2020discoveryofa pages 1-2
15. liu2020chemicalbiologytoolkit pages 10-11
16. luo2023doublecortinlikekinase1 pages 13-14
17. patel2016biochemicalandstructural pages 1-3
18. patel2016biochemicalandstructural pages 3-4
19. rogers2020autoregulatorycontrolof pages 1-3
20. rogers2021autoregulatorycontrolof pages 1-2
21. rogers2021autoregulatorycontrolof pages 2-4
22. rogers2021autoregulatorycontrolof pages 20-21
23. venkat2023mechanisticandevolutionary pages 16-17
24. venkat2023mechanisticandevolutionary pages 18-19
25. venkat2023mechanisticandevolutionary pages 22-23
26. venkat2023mechanisticandevolutionary pages 23-24
27. venkat2023mechanisticandevolutionary pages 24-25

References

1. (carli2022thefunctionof pages 157-160): ALE Carli. The function of doublecortin-like kinase 1 (dclk1) in gastric cancer. Unknown journal, 2022.
2. (carli2022thefunctionof pages 160-180): ALE Carli. The function of doublecortin-like kinase 1 (dclk1) in gastric cancer. Unknown journal, 2022.
3. (carli2022thefunctionof pages 210-213): ALE Carli. The function of doublecortin-like kinase 1 (dclk1) in gastric cancer. Unknown journal, 2022.
4. (carli2022thefunctionof pages 248-250): ALE Carli. The function of doublecortin-like kinase 1 (dclk1) in gastric cancer. Unknown journal, 2022.
5. (carli2022thefunctionof pages 38-42): ALE Carli. The function of doublecortin-like kinase 1 (dclk1) in gastric cancer. Unknown journal, 2022.
6. (carli2022thefunctionofa pages 160-180): ALE Carli. The function of doublecortin-like kinase 1 (dclk1) in gastric cancer. Unknown journal, 2022.
7. (carli2022thefunctionofa pages 210-213): ALE Carli. The function of doublecortin-like kinase 1 (dclk1) in gastric cancer. Unknown journal, 2022.
8. (carli2023structureguidedpredictionof pages 1-2): Annalisa L. E. Carli, Joshua M. Hardy, Hanadi Hoblos, Matthias Ernst, Isabelle S. Lucet, and Michael Buchert. Structure-guided prediction of the functional impact of dclk1 mutations on tumorigenesis. Biomedicines, 11:990, Mar 2023. URL: https://doi.org/10.3390/biomedicines11030990, doi:10.3390/biomedicines11030990. This article has 2 citations and is from a peer-reviewed journal.
9. (carli2023structureguidedpredictionof pages 16-17): Annalisa L. E. Carli, Joshua M. Hardy, Hanadi Hoblos, Matthias Ernst, Isabelle S. Lucet, and Michael Buchert. Structure-guided prediction of the functional impact of dclk1 mutations on tumorigenesis. Biomedicines, 11:990, Mar 2023. URL: https://doi.org/10.3390/biomedicines11030990, doi:10.3390/biomedicines11030990. This article has 2 citations and is from a peer-reviewed journal.
10. (carli2023structureguidedpredictionof pages 19-20): Annalisa L. E. Carli, Joshua M. Hardy, Hanadi Hoblos, Matthias Ernst, Isabelle S. Lucet, and Michael Buchert. Structure-guided prediction of the functional impact of dclk1 mutations on tumorigenesis. Biomedicines, 11:990, Mar 2023. URL: https://doi.org/10.3390/biomedicines11030990, doi:10.3390/biomedicines11030990. This article has 2 citations and is from a peer-reviewed journal.
11. (carli2023structureguidedpredictionof pages 2-4): Annalisa L. E. Carli, Joshua M. Hardy, Hanadi Hoblos, Matthias Ernst, Isabelle S. Lucet, and Michael Buchert. Structure-guided prediction of the functional impact of dclk1 mutations on tumorigenesis. Biomedicines, 11:990, Mar 2023. URL: https://doi.org/10.3390/biomedicines11030990, doi:10.3390/biomedicines11030990. This article has 2 citations and is from a peer-reviewed journal.
12. (carli2023structureguidedpredictionof pages 4-6): Annalisa L. E. Carli, Joshua M. Hardy, Hanadi Hoblos, Matthias Ernst, Isabelle S. Lucet, and Michael Buchert. Structure-guided prediction of the functional impact of dclk1 mutations on tumorigenesis. Biomedicines, 11:990, Mar 2023. URL: https://doi.org/10.3390/biomedicines11030990, doi:10.3390/biomedicines11030990. This article has 2 citations and is from a peer-reviewed journal.
13. (carli2023structureguidedpredictionof pages 8-9): Annalisa L. E. Carli, Joshua M. Hardy, Hanadi Hoblos, Matthias Ernst, Isabelle S. Lucet, and Michael Buchert. Structure-guided prediction of the functional impact of dclk1 mutations on tumorigenesis. Biomedicines, 11:990, Mar 2023. URL: https://doi.org/10.3390/biomedicines11030990, doi:10.3390/biomedicines11030990. This article has 2 citations and is from a peer-reviewed journal.
14. (ferguson2020discoveryofa pages 1-2): F. Ferguson, Behnam Nabet, Srivatsan Raghavan, Srivatsan Raghavan, Yan Liu, Alan L. Leggett, Miljan Kuljanin, R. Kalekar, R. Kalekar, Annan Yang, Annan Yang, Shuning He, Jinhua Wang, Raymond W.S. Ng, Raymond W.S. Ng, Rita Sulahian, Lianbo Li, Emily J Poulin, Ling Huang, Jošt Vrabič Koren, Nora Diéguez-Martínez, Sergio Espinosa, Zhiyang Zeng, Cesear R. Corona, J. Vasta, R. Ohi, Taebo Sim, N. Kim, W. Harshbarger, W. Harshbarger, J. Lizcano, M. Robers, Senthil Muthaswamy, Charles Y. Lin, A. Look, K. Haigis, J. Mancias, B. Wolpin, Andrew J. Aguirre, Andrew J. Aguirre, William C. Hahn, William C. Hahn, K. Westover, and N. Gray. Discovery of a selective inhibitor of doublecortin like kinase 1. Nature Chemical Biology, 16:635-643, Apr 2020. URL: https://doi.org/10.1038/s41589-020-0506-0, doi:10.1038/s41589-020-0506-0. This article has 100 citations and is from a highest quality peer-reviewed journal.
15. (liu2020chemicalbiologytoolkit pages 10-11): Yan Liu, Fleur M. Ferguson, Lianbo Li, Miljan Kuljanin, Caitlin E. Mills, Kartik Subramanian, Wayne Harshbarger, Sudershan Gondi, Jinhua Wang, Peter K. Sorger, Joseph D. Mancias, Nathanael S. Gray, and Kenneth D. Westover. Chemical biology toolkit for dclk1 reveals connection to rna processing. Cell Chemical Biology, 27:1229-1240.e4, Oct 2020. URL: https://doi.org/10.1016/j.chembiol.2020.07.011, doi:10.1016/j.chembiol.2020.07.011. This article has 22 citations and is from a domain leading peer-reviewed journal.
16. (luo2023doublecortinlikekinase1 pages 13-14): Wu Luo, Yiyi Jin, Yuchen Jiang, Libin Yang, Haowen Xu, Di Wu, Yanmei Zhang, Lina Yin, Zia Ali Khan, Guang Liang, and Yi Wang. Doublecortin-like kinase 1 activates nf-κb to induce inflammatory responses by binding directly to ikkβ. Cell Death & Differentiation, 30:1184-1197, Mar 2023. URL: https://doi.org/10.1038/s41418-023-01147-8, doi:10.1038/s41418-023-01147-8. This article has 10 citations.
17. (patel2016biochemicalandstructural pages 1-3): Onisha Patel, Weiwen Dai, Mareike Mentzel, Michael D.W. Griffin, Juliette Serindoux, Yoann Gay, Stefanie Fischer, Shoukat Sterle, Ashleigh Kropp, Christopher J. Burns, Matthias Ernst, Michael Buchert, and Isabelle S. Lucet. Biochemical and structural insights into doublecortin-like kinase domain 1. Structure, 24 9:1550-61, Sep 2016. URL: https://doi.org/10.1016/j.str.2016.07.008, doi:10.1016/j.str.2016.07.008. This article has 65 citations and is from a domain leading peer-reviewed journal.
18. (patel2016biochemicalandstructural pages 3-4): Onisha Patel, Weiwen Dai, Mareike Mentzel, Michael D.W. Griffin, Juliette Serindoux, Yoann Gay, Stefanie Fischer, Shoukat Sterle, Ashleigh Kropp, Christopher J. Burns, Matthias Ernst, Michael Buchert, and Isabelle S. Lucet. Biochemical and structural insights into doublecortin-like kinase domain 1. Structure, 24 9:1550-61, Sep 2016. URL: https://doi.org/10.1016/j.str.2016.07.008, doi:10.1016/j.str.2016.07.008. This article has 65 citations and is from a domain leading peer-reviewed journal.
19. (rogers2020autoregulatorycontrolof pages 1-3): Melissa M. Rogers, Amrita Ramkumar, Ashlyn M. Downing, Hannah Bodin, Julia Castro, Dan W. Nowakowski, and Kassandra M. Ori-McKenney. Autoregulatory control of microtubule binding in the oncogene, doublecortin-like kinase 1. BioRxiv, Jun 2020. URL: https://doi.org/10.1101/2020.06.12.149252, doi:10.1101/2020.06.12.149252. This article has 1 citations.