1. Phylogeny  
   Serine/threonine‐protein kinase DCLK1 (also known as DCAMKL1, DCDC3A, or KIAA0369; UniProt: O15075) is a member of the doublecortin (DCX) family that is evolutionarily conserved among metazoans. DCLK1 falls within the larger calcium/calmodulin‐dependent kinase (CAMK) group and shares conserved sequence motifs and domain architecture with kinases involved in neuronal migration and microtubule regulation. Orthologs of DCLK1 are found across mammalian species and other eukaryotes, and gene duplication events have produced related paralogs such as DCLK2 and DCLK3. In addition, alternative splicing generates isoforms (for example, DCLK1.1 and DCLK1.2) that differ primarily in the sequence and length of their C‐terminal regulatory tail, which modulates autoinhibition. These evolutionary relationships and isoform‐specific variations underscore a conserved kinase core accompanied by unique regulatory elements that have emerged during eukaryotic evolution (venkat2023mechanisticandevolutionary pages 1-2, dijkmans2010thedoublecortingene pages 10-11).
2. Reaction Catalyzed  
   DCLK1 catalyzes the phosphorylation reaction in which ATP and a protein substrate containing serine or threonine are converted to ADP, a phosphorylated protein, and a proton. This reaction can be summarized as:  
     ATP + [protein]‑(L‑serine or L‑threonine) → ADP + [protein]‑(L‑serine/threonine)‑phosphate + H⁺  
   This chemical transformation is typical for serine/threonine kinases and is central to the regulation of numerous cellular processes (carli2022thefunctionofa pages 257-259).
3. Cofactor Requirements  
   The catalytic activity of DCLK1 is dependent on the presence of divalent metal ions. In common with many serine/threonine kinases, DCLK1 requires Mg²⁺ as a cofactor for optimal ATP binding and phosphoryl transfer. This Mg²⁺ dependency is necessary to stabilize the negative charges of ATP’s phosphate groups during catalysis (patel2016biochemicalandstructural pages 3-4, venkat2023mechanisticandevolutionary pages 1-2).
4. Substrate Specificity  
   DCLK1 is a serine/threonine kinase with substrate specificity that, based on recent substrate‐specificity profiling studies, favors certain consensus motifs typical of the human serine/threonine kinome. In addition to its autophosphorylation capabilities, proteomic and phosphoproteomic studies have revealed that DCLK1 modulates phosphorylation on several substrates involved in cytoskeletal regulation, RNA processing, autophagy, and cell cycle control. In particular, phosphorylation sites identified in substrates such as RPS6KA3, ATG9A, CMTR1, BIRC3, and CDC42BPG suggest that DCLK1 recognizes and phosphorylates sequences with specific amino acid preferences. Recent work (Johnson et al. 2023) has provided an atlas of substrate specificities for the human serine/threonine kinome, and although the precise consensus motif for DCLK1 is still under active investigation, it appears to require a local environment that may include hydrophobic and basic residues proximal to the phosphorylation site (carli2022thefunctionofa pages 150-154, carli2023structureguidedpredictionof pages 16-17).
5. Structure  
   DCLK1 is a multidomain protein characterized by a modular organization that underpins its dual functions as both a microtubule‐associated protein (MAP) and a catalytic kinase. The N-terminus comprises two tandem doublecortin (DC) domains—designated DC1 and DC2—that share approximately 80% and 87% sequence similarity, respectively, with the classical DCX protein. DC1 preferentially binds to polymerized microtubules (α/β-tubulin heterodimers), whereas DC2 has the capacity to interact with both polymerized and soluble tubulin, thereby facilitating microtubule nucleation and stabilization (carli2022thefunctionof pages 42-45, carli2022thefunctionof pages 45-48; dijkmans2010thedoublecortingene pages 10-11).

Connecting the DC domains to its catalytic domain is a PEST-rich linker region that is enriched in proline, glutamic acid, serine, and threonine residues. This region is thought to be highly accessible on the protein surface and may act as a docking platform for other regulatory proteins or proteolytic enzymes such as calpain. Following the PEST linker, the C-terminal portion of DCLK1 contains a serine/threonine kinase domain that adopts a canonical bilobal structure comprising an N-terminal lobe responsible for ATP binding and a larger C-terminal lobe responsible for substrate recognition. Within the kinase domain, key elements including the glycine-rich loop, the catalytic loop (featuring the invariant D511 residue critical for catalysis), the DFG motif in the activation segment, and the C-helix (which participates in a critical salt bridge with a lysine residue) have been identified by crystallographic studies and are consistent with a CaMKI-like fold (patel2016biochemicalandstructural pages 1-3, carli2022thefunctionof pages 42-45).

The C-terminal tail of DCLK1 functions as an autoinhibitory domain (AID) that modulates kinase activity. Structural models, including those derived from AlphaFold predictions, suggest that this tail makes intramolecular contacts with the catalytic domain, occluding the ATP-binding site and thereby suppressing kinase activity in the absence of appropriate activating signals. A well-known kinase-dead mutation, D511N, serves as an important functional marker by abolishing catalytic activity and simultaneously altering microtubule binding, underscoring the interdependence of the kinase and microtubule-regulatory functions (carli2022thefunctionof pages 42-45, carli2022thefunctionof pages 45-48, patel2016biochemicalandstructural pages 3-4).

1. Regulation  
   DCLK1 is regulated at multiple levels through post-translational modifications as well as intramolecular interactions that control the balance between its kinase activity and microtubule binding. Autophosphorylation is a key regulatory mechanism whereby DCLK1 phosphorylates itself on specific serine residues, including those within or near the DC domains. In particular, phosphorylation at Ser32—a residue homologous to Ser28 in DCX—acts as a molecular switch that modulates DCLK1’s affinity for microtubules, with phosphorylation leading to reduced microtubule binding and enhanced actin interaction (carli2022thefunctionof pages 42-45, carli2022thefunctionofa pages 150-154, rogers2020autoregulatorycontrolof pages 1-3).

The PEST-rich linker region is heavily phosphorylated by multiple kinases including CDK5, GSK3, ERK1, and JNK; these modifications influence cellular localization and microtubule polymerization dynamics. This region may also serve as a site for proteolytic cleavage, further regulating the availability of distinct isoforms with different functional properties (carli2022thefunctionof pages 42-45, carli2022thefunctionof pages 45-48). In addition, the C-terminal autoinhibitory domain exerts negative regulation on the kinase domain by physically interfering with substrate and ATP access; removal of this domain or mutations that destabilize its interaction (such as D511N) results in derepression of kinase activity and altered intracellular distribution (carli2022thefunctionofb pages 45-48, rogers2020autoregulatorycontrolof pages 3-5).

Upstream regulators, including kinases such as GSK3B and MARK1, have been implicated in modulating phosphorylation within the N-terminal regions of DCLK1. Moreover, there is evidence that DCLK1 activity may influence adjacent phosphatases (for example ILKAP) that indirectly regulate phosphorylation of its own regulatory sites. These convergent mechanisms contribute to a highly dynamic and context-dependent regulation of DCLK1 in cellular pathways (carli2022thefunctionof pages 210-213, rogers2020autoregulatorycontrolof pages 1-3).

1. Function  
   DCLK1 is a multifunctional protein that integrates kinase signaling with microtubule dynamics, thus contributing to both neuronal development and cellular homeostasis. In the developing brain, DCLK1 is implicated in calcium-signaling pathways that control neuronal migration, thereby affecting the proper formation of cortical layers. Its prominent expression in neuronal progenitor cells and migrating neurons supports a role in guiding axon outgrowth, dendritic branching, and cellular polarity during neurogenesis (carli2022thefunctionofa pages 38-42, carli2022thefunctionofa pages 45-48).

Beyond its roles in the nervous system, DCLK1 is overexpressed in several solid tumors, including gastric, colorectal, pancreatic, and renal cancers, where it correlates with poor clinical outcomes. In these settings, DCLK1 functions as a marker of cancer stem cells (CSCs) and is implicated in promoting epithelial-to-mesenchymal transition (EMT), cell invasion, and metastasis. Proteomic analyses have identified an array of potential substrates whose phosphorylation by DCLK1 modulates cytoskeletal reorganization, cell cycle progression, and extracellular matrix remodeling. These substrates include proteins involved in MAPK signaling (e.g., RPS6KA3), autophagy (e.g., ATG9A), anti-apoptotic pathways (e.g., BIRC3), and RNA processing (e.g., CMTR1) (carli2022thefunctionof pages 160-180, carli2022thefunctionofb pages 210-213).

DCLK1’s dual capacity to function as a microtubule-binding MAP and a catalytic kinase ensures that it can coordinate intracellular transport, mitotic spindle organization, and cell polarity. This coordination is essential during cell division and migration and provides a mechanistic link between structural dynamics and signal transduction. The aberrant expression and activity of DCLK1 in malignant cells contribute to dysregulated cell motility and invasiveness, in part by altering the phosphorylation state of key cytoskeletal regulators (carli2022thefunctionof pages 38-42, carli2022thefunctionofa pages 210-213).

1. Other Comments  
   Several small-molecule inhibitors targeting DCLK1 have been developed, most notably DCLK1‑IN‑1, which has demonstrated potent and selective inhibition of DCLK1 kinase activity in preclinical models. These inhibitors are being investigated for their potential to reverse oncogenic phenotypes, particularly in gastrointestinal cancers. In addition, chemical biology toolkits have been established that include engineered kinase-dead mutants (such as D511N) to dissect DCLK1 function and to validate inhibitor specificity (ferguson2020discoveryofa pages 7-8, liu2020chemicalbiologytoolkit pages 10-11).

DCLK1 is also associated with altered vesicular trafficking and extracellular vesicle composition in tumor cells, which may contribute to changes in the tumor microenvironment and promote metastatic behavior. In several cancer studies, DCLK1 overexpression correlates with enhanced EMT and poor overall survival, underscoring its potential as a diagnostic marker and therapeutic target. In the context of neurobiology, DCLK1 participates in key calcium-signaling pathways that are critical for normal brain development and cellular homeostasis, suggesting that its dysregulation may also contribute to neurological disorders.

Notable disease mutations affecting DCLK1 have not been described in detail in the current context; however, the kinase-dead mutation D511N is frequently used as a functional inactivation control in experimental systems. The interplay between DCLK1’s kinase activity and its microtubule-binding function is central to its role in both normal development and disease, and ongoing studies continue to refine our understanding of its regulatory mechanisms via phosphorylation and proteolytic processing (chen2023molecularmechanismof pages 15-17).

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