1. Phylogeny  
   Casein kinase I isoform delta (CK1δ), encoded by the CSNK1D gene and identified with UniProt ID P48730, is a member of the casein kinase 1 (CK1) family of serine/threonine kinases that is evolutionarily ancient and ubiquitously expressed in eukaryotic cells (bischof2013ck1δkinaseactivity pages 8-9). Evolutionary reconstructions indicate that the catalytic domains of CK1 family members are highly conserved across species, reflecting an origin that can be traced back to the Last Eukaryotic Common Ancestor (LECA) (bischof2013ck1δkinaseactivity pages 9-12). In yeast, orthologous proteins such as HRR25 and members of the YCK family exhibit significant similarity with mammalian CK1δ, confirming the conservation of key structural motifs such as the glycine-rich P-loop and the catalytic lysine residue (knippschild2005theroleof pages 1-2). Comparative analyses have shown that the CK1 enzyme family, including CK1δ, consistently groups with the serine/threonine kinases when mapping the human kinome, and its catalytic core is maintained in organisms ranging from unicellular yeasts to complex mammals (meng2019kinaseactivityof pages 1-2). Mammalian CK1δ is most closely related to CK1ε, and despite nearly identical kinase domains, they are distinguished by differences in their regulatory C-terminal tails that provide isoform-specific modulation (bischof2013ck1δkinaseactivity pages 8-9, bischof2013ck1δkinaseactivity pages 9-12). Alternative splicing of CSNK1D transcripts results in isoforms such as CK1δ1 and CK1δ2; these variants differ largely in their disordered C-terminal regulatory regions, which in turn contribute to discrete subcellular localizations and regulatory properties (bischof2013ck1δkinaseactivity pages 9-12). Further, phylogenetic studies underscore that the kinase domain of CK1δ contains conserved features—including a defined P-loop, a catalytic loop with an essential aspartate residue, and a conserved lysine required for ATP binding—across different eukaryotes (xu2019structureregulationand pages 3-4). These observations collectively reinforce the notion that CK1δ is an evolutionarily conserved enzyme with a central role in the regulation of cellular signaling pathways (venerando2022editorialcaseinkinases pages 1-3).
2. Reaction Catalyzed  
   CK1δ functions as an ATP-dependent serine/threonine protein kinase that catalyzes the phosphoryl transfer reaction essential for numerous signal transduction processes (perez2011proteinkinasesck1 pages 1-3). In its catalytic cycle, CK1δ binds ATP and positions it precisely in its active site so that the terminal (γ) phosphate group is transferred to the hydroxyl group of a serine or threonine residue on the substrate protein (eng2017sitespecificphosphorylationof pages 8-10). The overall chemical reaction can be summarized by the following equation:  
     ATP + [protein]–(L‑serine or L‑threonine) → ADP + [protein]–(L‑serine/threonine)-phosphate + H⁺  
   This reaction depends on the proper orientation of the substrate within the catalytic cleft, where conserved residues such as Lys-38 secure the positioning of ATP and facilitate the nucleophilic attack by the substrate’s hydroxyl group (bischof2013ck1δkinaseactivity pages 9-12). Thus, CK1δ’s phosphoryl transfer activity plays a fundamental role in modulating the function, activity, and interactions of a myriad of substrate proteins across various cellular contexts (perez2011proteinkinasesck1 pages 1-3).
3. Cofactor Requirements  
   The catalytic activity of CK1δ is strictly dependent on the presence of divalent metal ions, with magnesium (Mg²⁺) being the most critical cofactor (perez2011proteinkinasesck1 pages 1-3). Mg²⁺ ions bind in the active site of CK1δ along with ATP, where they neutralize the negative charges of the phosphate groups and stabilize the ATP–kinase complex (meng2019kinaseactivityof pages 1-2). This stabilization is essential for the correct alignment of ATP with key catalytic residues, including the conserved lysine that interacts with ATP and the aspartate residue within the catalytic loop that acts as a base (xu2019structureregulationand pages 4-6). The necessity for Mg²⁺ is a common feature among serine/threonine kinases and is fundamental to the phosphoryl transfer mechanism employed by CK1δ (xu2019structureregulationand pages 4-6).
4. Substrate Specificity  
   CK1δ exhibits a distinct substrate specificity that is largely determined by the amino acid sequence context surrounding the target serine or threonine residue (eng2017sitespecificphosphorylationof pages 8-10). The enzyme preferentially phosphorylates substrates that contain either a cluster of acidic residues or a “primed” phosphorylated serine/threonine residue located three to four amino acids upstream of the target site (perez2011proteinkinasesck1 pages 1-3). In many cases, this requirement results in the recognition of a canonical consensus motif characterized by a phospho-primed residue or an intrinsic acidic character, which enhances substrate binding through electrostatic interactions with positively charged residues in the CK1δ active site such as Arg-178 and Lys-224 (narasimamurthy2018ck1δεproteinkinase pages 2-2, narasimamurthy2018ck1δεproteinkinase pages 3-4). Additionally, alternative substrate motifs such as the SLS (Ser-Leu-Ser) motif have been identified in key targets like β-catenin, where the presence of acidic clusters further promotes efficient docking and subsequent phosphorylation (eng2017sitespecificphosphorylationof pages 8-10). Consequently, CK1δ is capable of modifying a broad range of substrates, from circadian regulators like PER2 to proteins involved in cell adhesion, signal transduction, and transcriptional regulation (perez2011proteinkinasesck1 pages 1-3).
5. Structure  
   The three-dimensional structure of CK1δ is defined by a canonical bilobal kinase domain that is characteristic of eukaryotic protein kinases (xu2019structureregulationand pages 3-4). The smaller N-terminal lobe primarily comprises β-strands and features a glycine-rich loop (or P-loop), with a conserved motif (for example, GSGSFG) that forms a “ceiling” over the ATP-binding pocket; this structural element is critical for proper positioning and binding of ATP (xu2019structureregulationand pages 3-4). In the N-lobe, a key lysine residue (Lys-38 in CK1δ) is highly conserved and plays an essential role in coordinating the phosphate groups of ATP, which is indispensable for the phosphoryl transfer reaction (bischof2013ck1δkinaseactivity pages 12-13). The larger C-terminal lobe is predominantly α-helical and encompasses several important catalytic features, including the activation loop (T-loop) that undergoes conformational rearrangements upon phosphorylation and contains a catalytic aspartate residue (Asp-128) that serves as the catalytic base during the reaction (xu2019structureregulationand pages 9-11). In addition to these structured regions, CK1δ possesses an intrinsically disordered C-terminal tail that is subject to extensive post-translational modifications, notably autophosphorylation; this flexible regulatory region modulates the enzyme’s basal activity by acting as an autoinhibitory domain when phosphorylated (bischof2013ck1δkinaseactivity pages 13-14, xu2019structureregulationand pages 24-25). Alternative splicing of the CSNK1D transcript gives rise to isoforms—such as CK1δ1 and CK1δ2—that differ primarily in the sequence and length of their C-terminal tails, thereby influencing autoinhibition, substrate docking, and subcellular localization (bischof2013ck1δkinaseactivity pages 9-12, xu2019structureregulationand pages 24-25). High-resolution insights from crystallographic studies and computational models (including AlphaFold predictions) reveal that the kinase domain of CK1δ is organized around a hydrophobic spine that stabilizes the active conformation, and a critical C-helix that aligns catalytic residues for efficient phosphoryl transfer (xu2019structureregulationand pages 3-4, xu2019structureregulationand pages 9-11). Moreover, structural data suggest that in certain regulatory contexts, CK1δ can form dimers through interactions mediated by regions in the C-terminal domain, potentially modulating ATP accessibility and catalytic efficiency (bischof2013ck1δkinaseactivity pages 12-13, bischof2013ck1δkinaseactivity pages 4-5). Together, these structural features not only facilitate the catalytic activity of CK1δ but also enable the integration of diverse regulatory inputs that determine substrate specificity and enzyme activity.
6. Regulation  
   The regulation of CK1δ is multifaceted and comprises intrinsic autophosphorylation, extrinsic phosphorylation by other kinases, as well as modulation through protein–protein interactions and conformational changes (eng2017sitespecificphosphorylationof pages 16-17). One of the primary regulatory mechanisms involves autophosphorylation of multiple serine and threonine residues located within the intrinsically disordered C-terminal tail; these post-translational modifications generate inhibitory phosphate groups that act as pseudo-substrates, thereby reducing the basal kinase activity (bischof2013ck1δkinaseactivity pages 13-14). In addition to autophosphorylation, CK1δ is phosphorylated in trans by several upstream kinases. For example, checkpoint kinase 1 (Chk1) phosphorylates specific residues within the regulatory domain of CK1δ, resulting in an approximate 30% reduction in its activity (bischof2013ck1δkinaseactivity pages 9-12). Protein kinase C alpha (PKCα) also phosphorylates CK1δ, targeting residues within both the catalytic and regulatory regions, which in turn modulate substrate binding and overall enzyme turnover (meng2019kinaseactivityof pages 1-2). Moreover, cyclin-dependent kinases (CDKs) have been implicated in phosphorylating threonine residues in the C-terminal tail (such as T344 and T347), modifications that are particularly relevant in circadian regulation where altered phosphorylation of clock proteins like PER2 is observed (eng2017sitespecificphosphorylationof pages 16-17, bischof2013ck1δkinaseactivity pages 9-12). The reversible nature of these phosphorylation events is maintained by protein phosphatases, notably PP1, which dephosphorylate CK1δ, restoring its activity when appropriate (eng2017sitespecificphosphorylationof pages 16-17). Furthermore, allosteric interactions between the phosphorylated C-terminal tail and the kinase domain provide an additional layer of regulation, wherein conformational changes induced by substrate binding or interactions with regulatory proteins shift CK1δ between active and inactive states (harold2023caseinkinase1δ pages 28-34, xu2019structureregulationand pages 24-25). The existence of alternatively spliced variants with distinct C-terminal tails (e.g., CK1δ1 versus CK1δ2) further refines the enzyme’s regulatory landscape by affecting both autophosphorylation efficiency and subcellular localization (bischof2013ck1δkinaseactivity pages 9-12). Collectively, these diverse regulatory mechanisms enable CK1δ to function as a finely tuned molecular switch, capable of integrating multiple cellular signals to modulate its catalytic activity in a context-dependent manner.
7. Function  
   CK1δ is a central regulator of multiple fundamental cellular processes and exerts its effects through the phosphorylation of a broad spectrum of substrate proteins (perez2011proteinkinasesck1 pages 1-3). One of its primary roles is in the control of circadian rhythms, where CK1δ phosphorylates core clock proteins such as PER1 and PER2; these phosphorylation events govern the stability, nuclear transport, and proteasome-mediated degradation of the clock proteins, thus determining the period and amplitude of circadian oscillations (eng2017sitespecificphosphorylationof pages 8-10, perez2011proteinkinasesck1 pages 1-3). In addition, CK1δ is involved in the regulation of Wnt signaling pathways through its action on Dishevelled proteins (DVL2 and DVL3), a critical process for modulating gene expression programs that control cell proliferation and embryonic development (perez2011proteinkinasesck1 pages 1-3). The enzyme also phosphorylates connexin-43 (GJA1), which is essential for the formation and function of gap junctions, thereby influencing cell–cell communication and tissue homeostasis (perez2011proteinkinasesck1 pages 1-3). Beyond these roles, CK1δ contributes to the maintenance of genomic integrity by phosphorylating key proteins involved in the DNA damage response and cell cycle control, such as the tumor suppressor p53/TP53 and topoisomerase IIα (TOP2A) (perez2011proteinkinasesck1 pages 1-3). Phosphorylation of p53 by CK1δ can influence cell cycle arrest and apoptosis, which are vital for safeguarding cells against genotoxic stress (perez2011proteinkinasesck1 pages 1-3). Furthermore, CK1δ has been shown to regulate transcriptional coactivators such as estrogen receptor (ESR1) and AIB1/NCOA3, thereby modulating transcriptional activity and impacting cell growth and survival (perez2011proteinkinasesck1 pages 1-3). The kinase also phosphorylates DNMT1, which leads to a reduction in the DNA-binding capacity of this enzyme, potentially affecting epigenetic regulation (perez2011proteinkinasesck1 pages 1-3). Additionally, CK1δ-mediated phosphorylation of YAP1 promotes its recognition by the SCF(β-TRCP) E3 ubiquitin ligase complex, linking CK1δ to the regulation of the Hippo signaling pathway and tissue growth control (perez2011proteinkinasesck1 pages 1-3). The wide substrate repertoire of CK1δ, encompassing proteins involved in circadian clock regulation, signal transduction, cell communication, and genomic stability, underscores its multifunctional role in maintaining cellular homeostasis across diverse tissues including the nervous system, liver, muscle, and connective tissue.
8. Other Comments  
   Given its pivotal role in a multitude of cellular pathways, CK1δ has emerged as an attractive target for therapeutic intervention in several disease contexts. Small-molecule inhibitors that target the ATP-binding pocket or interfere with the autoinhibitory interactions of the C-terminal tail have been developed and are being explored for their potential in treating conditions where aberrant CK1δ activity is implicated (cescon2023reversibleandcovalent pages 43-48). Notably, dysregulation of CK1δ activity is associated with neurodegenerative disorders such as Alzheimer’s disease, where abnormal tau phosphorylation plays a contributory role, as well as with various cancers where CK1δ-mediated phosphorylation of substrates like p53 influences tumor progression (perez2011proteinkinasesck1 pages 1-3, cescon2023reversibleandcovalent pages 43-48). The existence of alternatively spliced variants, such as CK1δ1 and CK1δ2, adds an additional layer of complexity; these isoforms differ in their C-terminal regulatory regions, which can affect both their autoinhibitory potency and subcellular distribution, and may serve as the basis for developing isoform-specific inhibitors or diagnostic tools (bischof2013ck1δkinaseactivity pages 9-12, harold2023caseinkinase1δ pages 28-34). Moreover, post-translational modifications such as phosphorylation by upstream kinases—including Chk1, PKCα, and CDKs—are critical for CK1δ regulation and have been shown to modulate its activity toward key substrates like PER2, thereby impacting circadian timing and other regulatory processes (eng2017sitespecificphosphorylationof pages 16-17, bischof2013ck1δkinaseactivity pages 9-12). In addition to chemical inhibitors like IC261 and PF-670462, which have been documented to decrease CK1δ activity in cell-based models, ongoing research is further defining the inhibitor profiles and selectivity for CK1δ in various pathological settings, including metabolic disorders and neurodegeneration (cescon2023reversibleandcovalent pages 43-48). These insights position CK1δ not only as a critical node in the regulation of cellular homeostasis but also as a highly promising drug target whose modulation may yield therapeutic benefits in a range of clinical conditions.
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