1. Phylogeny  
   Cyclin‐dependent kinase 10 (CDK10, gene: CDK10, UniProt: Q15131) is a member of the cyclin‐dependent kinase family that belongs to the CMGC group of kinases, a subgroup of the eukaryotic protein kinome conserved from yeast to mammals, although a yeast homologue is notably absent for CDK10 (duster2021biochemicalcharacterizationof pages 116-118). In evolutionary terms, CDK10 clusters with other transcription‐related CDKs and is phylogenetically more related to kinases such as CDK11 than to the classical cell cycle CDKs (e.g., CDK1, CDK2) (guen2013cdk10cyclinmis pages 1-1, guen2017theawakeningof pages 1-2). Its evolutionary origin can be traced to the expansion of the CDK family in metazoans, and its partnership with Cyclin M (also known as Cyclin Q; gene FAM58A) is conserved among higher organisms, highlighting an evolutionarily conserved regulatory module that is part of an ancient cell regulatory machinery (duster2021biochemicalcharacterizationofa pages 23-28, łukasik2021cyclindependentkinases(cdk) pages 5-7). Moreover, when compared with the global kinome described by Manning and co-workers, CDK10 falls into a subset of kinases with roles in transcription, reinforcing its distinct evolutionary trajectory from purely cell cycle‐regulatory kinases (Manning2002, Manning2002).
2. Reaction Catalyzed  
   CDK10 catalyzes the transfer of a γ-phosphate group from ATP to the hydroxyl side chain of serine or threonine residues in target proteins. The chemical reaction can be summarized as: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (duster2021biochemicalcharacterizationofa pages 86-89). This classical serine/threonine phosphorylation reaction is typical of members of the CDK family (colas2020cyclindependentkinasesand pages 1-2).
3. Cofactor Requirements  
   The kinase activity of CDK10 depends on the presence of ATP as the phosphate donor and requires magnesium ions (Mg²⁺) as an essential cofactor for catalysis (duster2021biochemicalcharacterizationofa pages 86-89). Mg²⁺ coordinates ATP binding in the active site and assists in proper orientation for effective phosphoryl transfer to the target substrates (duster2021biochemicalcharacterizationof pages 33-37).
4. Substrate Specificity  
   CDK10 exhibits substrate specificity that is in line with many serine/threonine kinases, primarily targeting serine/threonine residues that are followed by a proline residue in the substrate sequence. In vitro studies have demonstrated that CDK10 phosphorylates the transcription factor ETS2 on adjacent serine residues, leading to its proteasomal degradation (guen2013cdk10cyclinmis pages 1-2, bazzi2021cdk10ingastrointestinal pages 1-2). Furthermore, CDK10 phosphorylates regulators of actin cytoskeleton organization such as PKN2, thereby influencing actin dynamics and negatively regulating ciliogenesis through the modulation of RhoA signaling (bazzi2021cdk10ingastrointestinal pages 1-2, duster2021biochemicalcharacterizationofa pages 15-18). In addition to ETS2 and PKN2, CDK10 has been shown to modify other substrates including segments of the RNA polymerase II C-terminal domain (CTD) and the proto-oncogene c-Myc, with biochemical assays indicating a modest preference for the phosphorylation of serine residues within (S/T)P motifs (duster2021biochemicalcharacterizationof pages 89-93, guen2017theawakeningof pages 2-3). According to recent high-throughput kinase substrate profiling, such as that presented by Johnson et al. (2023), serine/threonine kinases frequently display substrate motifs enriched in basic residues at defined positions relative to the phosphoacceptor; thus, CDK10 is thought to share a consensus that is centered on a minimal (S/T)P motif with preferential flanking sequences that enhance its catalytic efficiency (Johnson2023). This pattern contrasts with tyrosine kinases whose intrinsic substrate recognition preferences have been separately characterized (Yaron-Barir2024).
5. Structure  
   CDK10 is a 360 amino acid protein characterized by the canonical bilobal structure of eukaryotic protein kinases, comprising an N-terminal lobe mainly involved in ATP binding and a larger C-terminal lobe that is primarily responsible for substrate recognition (duster2021biochemicalcharacterizationofa pages 86-89). A notable structural feature of CDK10 is its modified cyclin interaction motif “PISSLRE,” which is a variant of the canonical PSTAIRE motif found in other CDKs; this modification is essential for its specific interaction with Cyclin M (duster2021biochemicalcharacterizationofa pages 86-89, guen2013cdk10cyclinmis pages 1-2). Within the kinase domain, the catalytic loop contains a critical aspartate residue (Asp163) that acts as a proton acceptor during phosphoryl transfer, and mutation of this residue (e.g., D163N) abolishes catalytic activity, underscoring its central role in the enzyme’s function (duster2021biochemicalcharacterizationofa pages 86-89). In addition to the catalytic loop, the activation segment or T-loop contains a key threonine residue (Thr196) whose phosphorylation is required for full activation of CDK10, although the upstream kinase responsible for this modification has not been definitively identified (duster2021biochemicalcharacterizationofa pages 86-89). CDK10 also possesses a bipartite nuclear localization signal located at its C-terminus, a feature that directs the enzyme—or more specifically, the CDK10/Cyclin M complex—to the nucleus where many of its substrates are localized (duster2021biochemicalcharacterizationofa pages 86-89, duster2021biochemicalcharacterizationofa pages 11-15). Cyclin M, the activating subunit of CDK10, is a relatively small protein of approximately 248 amino acids that contains two cyclin box domains. Although Cyclin M lacks an intrinsic nuclear localization signal, its stable binding to CDK10 is essential for the correct subcellular distribution and catalytic competence of the complex (guen2013cdk10cyclinmis pages 1-2, duster2021biochemicalcharacterizationofa pages 33-37). Despite the absence of a high-resolution crystal structure for the CDK10/Cyclin M complex, biochemical and biophysical studies, including those employing fusion proteins and mutagenesis strategies, have provided a clear view of the overall domain organization and the key catalytic and regulatory features that govern CDK10 function (duster2021biochemicalcharacterizationof pages 33-37, sun2005homologymodelingand pages 1-2).
6. Regulation  
   The activity of CDK10 is principally regulated through its association with Cyclin M, which is required for its catalytic activity; CDK10 alone is catalytically inactive (guen2013cdk10cyclinmis pages 1-2, duster2021biochemicalcharacterizationofa pages 15-18). Phosphorylation plays a critical role in modulating CDK10 function: phosphorylation at threonine 196 within the activation loop is necessary for full catalytic activity, while additional phosphorylation events, such as those occurring at serine 351 when co-expressed with Cyclin M, may contribute to regulation of protein–protein interactions or stability (duster2021biochemicalcharacterizationofa pages 86-89, duster2021biochemicalcharacterizationof pages 33-37). In cellular contexts, CDK10 activity is further modulated by the proteasomal degradation pathways; for example, phosphorylation of its substrate ETS2 by the CDK10/Cyclin M complex facilitates subsequent ubiquitin-dependent degradation of ETS2 (guen2013cdk10cyclinmis pages 5-6). This process not only regulates the level of ETS2 but also influences downstream signaling cascades such as MAPK signaling (bazzi2021cdk10ingastrointestinal pages 1-2). In addition, regulatory mechanisms such as interactions with other factors (e.g., HSP90 and possibly the prolyl isomerase Pin1) may influence CDK10 stability and function, although the precise details of these interactions require further elucidation (duster2021biochemicalcharacterizationofa pages 112-116). Overall, the principal regulatory features of CDK10 include its dependency on Cyclin M for activation, the requirement for activating phosphorylation events on the T-loop (Thr196), and its ability to modulate substrate stability through phosphorylation-dependent degradation pathways (guen2013cdk10cyclinmis pages 5-6, guen2017theawakeningof pages 4-6).
7. Function  
   CDK10 exerts a multifaceted role in cellular function primarily through its kinase activity, which directs the phosphorylation of key regulatory proteins. One of the primary substrates of CDK10 is the ETS2 transcription factor; phosphorylation of ETS2 by CDK10 promotes its proteasomal degradation, thereby negatively regulating ETS2-dependent transcriptional programs and influencing downstream signaling pathways such as the MAPK cascade (bazzi2021cdk10ingastrointestinal pages 1-2, guen2013cdk10cyclinmis pages 5-6). In addition, CDK10 phosphorylates actin dynamics regulators, such as protein kinase N2 (PKN2), thereby participating in the regulation of actin cytoskeleton organization. This activity is linked to its role as a negative regulator of ciliogenesis because phosphorylation of PKN2 by the CDK10/Cyclin M complex promotes RhoA signaling, which in turn represses both the assembly and elongation of primary cilia (bazzi2021cdk10ingastrointestinal pages 1-2, duster2021biochemicalcharacterizationofa pages 15-18). Furthermore, CDK10 has been implicated in transcriptional regulation beyond its action on ETS2; substrates such as RNA polymerase II CTD and the oncoprotein c-Myc have been identified in biochemical assays, suggesting that CDK10 modulates processes related to gene expression and cell proliferation (duster2021biochemicalcharacterizationof pages 89-93, guen2017theawakeningof pages 2-3). CDK10 is expressed in several tissues and cell types, and its expression levels have been correlated with clinical parameters in gastrointestinal cancers where it may function either as a tumor suppressor or, in certain contexts, as an oncogene (bazzi2021cdk10ingastrointestinal pages 1-2, duster2022functionalcharacterizationof pages 1-2). In breast cancer cells, reduced expression of CDK10 is associated with tamoxifen resistance, highlighting its importance in hormone-dependent signaling pathways and therapeutic response (guen2013cdk10cyclinmis pages 5-6, guen2017theawakeningof pages 4-6). Thus, the functional repertoire of CDK10 encompasses regulation of transcription factor turnover, modulation of cytoskeletal dynamics, and control of cell cycle progression, all of which contribute to its roles in development and disease (robert2021functionalcharacterizationof pages 1-2).
8. Other Comments  
   Several experimental inhibitors developed for related CDKs have shown inhibitory activity against CDK10, although no inhibitor specific to CDK10 is yet approved for clinical use. ATP-competitive inhibitors such as Flavopiridol, Dinaciclib, and NVP-2 have been reported to inhibit CDK10 activity in biochemical assays; in particular, NVP-2 displays potent inhibition and shares overlapping inhibitor profiles with CDK9 (duster2021biochemicalcharacterizationof pages 93-97, robert2020developmentofa pages 10-10). In addition, recent high-throughput screening assays, including the homogeneous miniaturized kinase assay developed by Robert et al. (2020), have enabled the identification of first-generation small-molecule inhibitors targeting the CDK10/Cyclin M complex (robert2020developmentofa pages 1-2). CDK10 dysfunction is also implicated in developmental disorders; mutations affecting Cyclin M (FAM58A) that disrupt CDK10 complex formation are causative for STAR syndrome, an X-linked developmental disorder characterized by syndactyly, telecanthus, and renal malformations (guen2013cdk10cyclinmis pages 5-6, guen2017theawakeningof pages 7-8). Beyond developmental syndromes, altered CDK10 expression levels have been associated with cancer progression. For instance, reduced CDK10 expression in breast cancer cells correlates with increased ETS2 stability and enhanced MAPK signaling, contributing to drug resistance and poorer clinical outcomes (bazzi2021cdk10ingastrointestinal pages 1-2, guen2017theawakeningof pages 7-8). Future research may further clarify the kinase’s structure–function relationships and facilitate the development of more selective inhibitors that could be used to modulate its activity in disease contexts. Additionally, recent substrate specificity profiling efforts, as exemplified by studies on the human serine/threonine kinome (Johnson2023) and the human tyrosine kinome (Yaron-Barir2024), provide a framework that may be analogous for CDK10, emphasizing its preference for (S/T)P motifs and reinforcing its classification within the serine/threonine kinase category.
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