1. Phylogeny  
   Dual specificity tyrosine-phosphorylation‐regulated kinase 3 (DYRK3) is a member of the DYRK kinase family, which is subdivided into Class I (DYRK1A and DYRK1B) and Class II (DYRK2, DYRK3, and DYRK4) kinases. DYRK3, also known as regulatory erythroid kinase, shares close evolutionary relationships with its Class II counterparts and is a conserved protein across metazoans. Phylogenetic analyses indicate that DYRK3 and other DYRK family members belong to the CMGC group of serine/threonine kinases, a lineage that can be traced back to the last eukaryotic common ancestor, with orthologs observed in a broad range of species from yeast to mammals (kim2018crystalstructureof pages 1-6, boni2020thedyrkfamily pages 1-3, lindberg2021dualspecificitytyrosinephosphorylationregulated pages 1-2).
2. Reaction Catalyzed  
   DYRK3 catalyzes a phosphorylation reaction in which the γ‐phosphate from ATP is transferred to hydroxyl groups on serine and threonine residues of target substrate proteins. In addition to mediating substrate phosphorylation, DYRK3 undergoes an intramolecular autophosphorylation on a critical tyrosine residue in its activation loop that is necessary for full catalytic activation. Thus, the reaction can be summarized as:  
   ATP + [protein]–OH → ADP + [protein]–(pSer/pThr) + H⁺ (kim2018crystalstructureof pages 1-6).
3. Cofactor Requirements  
   The catalytic activity of DYRK3 is dependent on the presence of divalent metal ions, with Mg²⁺ serving as the essential cofactor that facilitates ATP binding and proper orientation of the substrate for phosphoryl transfer. Mg²⁺ requirement is a hallmark of most kinases in the CMGC group, including the DYRK family (kim2018crystalstructureof pages 6-9).
4. Substrate Specificity  
   DYRK3 functions as a dual‐specificity kinase by initially autophosphorylating a conserved tyrosine residue in its activation loop and subsequently phosphorylating several key substrates exclusively on serine and threonine residues. Although a precise consensus phosphorylation motif for DYRK3 has not been definitively determined, its substrate specificity aligns with the broader characteristics of DYRK family kinases, which generally preferentially phosphorylate substrates containing unstructured regions with serine/threonine residues. In mitosis, DYRK3-mediated phosphorylation targets include unstructured domains in proteins such as SRRM1 and PCM1, which participate in the regulation of membraneless organelle dynamics (kim2018crystalstructureof pages 6-9, moududee2024dualspecificitytyrosineregulatedkinases pages 7-9).
5. Structure  
   The three-dimensional architecture of DYRK3 comprises a central kinase domain that exhibits the classical bi-lobal fold characteristic of protein kinases. The N-terminal lobe is predominantly composed of β-sheets that facilitate ATP binding, while the C-terminal lobe is enriched in α-helices and houses the substrate binding site. Unique to Class II DYRKs, DYRK3 contains an N-terminal autophosphorylation accessory (NAPA) domain that is critical for masking hydrophobic surfaces and stabilizing the N-lobe, thereby facilitating proper intramolecular autophosphorylation. In addition, it features a conserved DYRK homology (DH) box located immediately upstream of the catalytic domain. Structural studies have revealed that DYRK3 harbors a characteristic insertion region, analogous to the MAP kinase insert found within the CMGC kinase family, that may contribute to substrate interactions and regulation. A key regulatory feature in the structure of DYRK3 is the phosphorylation at serine 350 (S350), which has been shown to enhance both the thermal stability and enzymatic activity of the kinase (kim2018crystalstructureof pages 1-6, kim2018crystalstructureof pages 6-9, kim2018crystalstructureof pages 18-22, kim2018crystalstructureof pages 22-31).
6. Regulation  
   DYRK3 regulation is governed primarily by autophosphorylation events that occur co-translationally and immediately following protein folding. The enzyme autophosphorylates a conserved tyrosine residue within its activation loop, an essential modification that triggers a conformational change allowing full serine/threonine kinase activity. Furthermore, phosphorylation at serine residue S350 contributes significantly to the stability and activity of the kinase, with mutation studies demonstrating reduced stability and decreased activity in the absence of proper phosphorylation at this site. The NAPA domain plays an additional regulatory role by ensuring correct folding and shielding hydrophobic regions in the N-lobe; its presence is indispensable for efficient autophosphorylation. Unlike other kinases that are activated by upstream regulatory enzymes, DYRK3’s activation occurs intrinsically, and its regulation is mediated by post-translational modifications rather than external kinase cascades (kim2018crystalstructureof pages 1-6, mercer2006mirkdyrk1bamultifunctional pages 10-12, moududee2024dualspecificitytyrosineregulatedkinases pages 5-7, lindberg2021dualspecificitytyrosinephosphorylationregulated pages 14-15).
7. Function  
   DYRK3 plays a critical role in cell cycle progression through its unique function as a dissolvase of membraneless organelles during mitosis. During the G2-to-M transition, DYRK3 facilitates the disassembly of stress granules, nuclear speckles, and pericentriolar material by phosphorylating multiple serine and threonine residues in the unstructured domains of target proteins such as SRRM1 and PCM1. This enzymatic activity is essential for the proper release of mitotic regulators, including proteins like ZNF207, ensuring smooth progression through mitosis following nuclear envelope breakdown. Beyond its role in organelle disassembly, DYRK3 is prominently expressed in hematopoietic cells, particularly late-stage erythroid progenitors, where its activity has been implicated in the attenuation of erythroblast development. Through these mechanisms, DYRK3 is thought to contribute to cellular homeostasis and has been associated with aberrations in hematopoietic processes, including potential contributions to anemic conditions (kim2018crystalstructureof pages 1-6, mercer2006mirkdyrk1bamultifunctional pages 1-2, boni2020thedyrkfamily pages 15-17, yoshida2023newinsightsinto pages 13-13).
8. Other Comments  
   DYRK3 has been reported to be inhibited by ATP-competitive inhibitors, with compounds such as harmine binding to its active site through hydrogen bonds and hydrophobic interactions. Research into broad-spectrum DYRK inhibitors has identified compound classes, including tetrahalobicycles, that inhibit DYRK3 activity at micromolar concentrations; however, selective inhibitors for DYRK3 are currently less well established. In the context of disease, the regulatory function of DYRK3 in disassembling membraneless organelles during mitosis and its role in modulating erythroblast development have attracted attention for their potential implications in cancer biology and hematopoietic disorders. Its dual-specificity activity and central regulatory role in cell cycle progression make DYRK3 an attractive candidate for further exploration as a therapeutic target, although detailed clinical associations remain to be fully elucidated (fernandezmartinez2015dyrk1athedoubleedged pages 7-8, schmitt2014designandsynthesis pages 17-18, boni2020thedyrkfamily pages 25-26).
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