1. Phylogeny  
   DYRK2 is a member of the dual‐specificity tyrosine‐phosphorylation‐regulated kinase (DYRK) family, a subgroup of the CMGC kinase superfamily that also includes Cyclin‐dependent kinases (CDKs), Mitogen-activated protein kinases (MAPKs), Glycogen synthase kinases (GSKs) and CDC-like kinases. Within mammals, the DYRK family comprises five members that are phylogenetically divided into two groups: Class I, which includes DYRK1A and DYRK1B, and Class II, which comprises DYRK2, DYRK3 and DYRK4. DYRK2, as the prototypical Class II member, is characterized by a high degree of conservation in its catalytic domain and is widely distributed among vertebrates, reflecting its early emergence during eukaryotic evolution. Comparative analyses indicate that orthologs of DYRK2 are present from invertebrates to mammals, underscoring its fundamental role in cell physiology. The conservation of sequence motifs, especially within the kinase domain and accessory regions, points to an evolutionary pressure to maintain its catalytic and regulatory functions across species (boni2020thedyrkfamily pages 1-3, yoshida2023newinsightsinto pages 2-4). Furthermore, sequence homology within the DYRK family suggests that DYRK2 shares common ancestral origins with other family members, with gene duplication events early in metazoan evolution leading to the divergence of Class I and Class II kinases. This phylogenetic context places DYRK2 as part of the core set of kinases that have been preserved since the Last Eukaryotic Common Ancestor (LECA) (tandon2021emergingrolesof pages 1-2).
2. Reaction Catalyzed  
   DYRK2 catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of serine or threonine residues on substrate proteins. In essence, the enzymatic reaction can be summarized as:  
     ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺.  
   In addition to phosphorylating exogenous substrates, DYRK2 undergoes an intramolecular autophosphorylation event on a key tyrosine residue within its activation loop during or immediately after translation. This autophosphorylation is indispensable for converting DYRK2 into its mature, fully active serine/threonine kinase form (correasaez2020updatingdualspecificitytyrosinephosphorylationregulated pages 1-2).
3. Cofactor Requirements  
   The catalytic function of DYRK2 depends on the presence of cofactors essential for phosphate transfer. As is typical for serine/threonine kinases, DYRK2 requires ATP as a phosphate donor. Moreover, the kinase activity of DYRK2 is dependent on divalent metal ions, with Mg²⁺ being the preferred cofactor that facilitates the proper positioning of ATP within the catalytic cleft and stabilizes the transition state during phosphorylation reactions (correasaez2020updatingdualspecificitytyrosinephosphorylationregulated pages 1-2, tandon2021emergingrolesof pages 11-12).
4. Substrate Specificity  
   DYRK2 exhibits substrate specificity that is defined by a consensus phosphorylation motif frequently encountered in its target proteins. Biochemical studies have shown that DYRK2 preferentially phosphorylates serine/threonine residues that are preceded, in many cases, by arginine residues at the −2 (or occasionally −3) position, and followed immediately by a proline residue at the +1 position. Thus, the consensus motif is generally described as Rxx(pS/T)P, where “pS/T” indicates the phosphorylated serine or threonine residue (correasaez2020updatingdualspecificitytyrosinephosphorylationregulated pages 4-7, correasaez2020updatingdualspecificitytyrosinephosphorylationregulated pages 7-9). This substrate signature underlies the ability of DYRK2 to function as a priming kinase in sequential phosphorylation cascades; for instance, phosphorylation of substrates such as c-Jun, c-Myc, and SNAIL often primes them for further phosphorylation by downstream kinases like GSK3β (correasaez2020updatingdualspecificitytyrosinephosphorylationregulated pages 14-15). In addition to these consensus sequences, DYRK2 phosphorylates key regulatory proteins such as p53, targeting Ser46, and modulates factors involved in cytoskeletal organization and metabolism, including NFATC1, EIF2B5, and GYS1, thereby refining its substrate specificity in response to cellular context (information section, boni2020thedyrkfamily pages 15-17).
5. Structure  
   DYRK2 possesses a modular architecture typical of protein kinases. Its structure can be divided into several distinct regions that contribute to its overall function. At the N-terminus, DYRK2 includes autophosphorylation accessory regions (often designated as NAPA domains) that facilitate cis-autophosphorylation events required for kinase activation (carmona2022determinationofnew pages 27-35). Adjacent to these regions, a nuclear localization signal (NLS) is embedded—approximately located around residues 189–191—thereby directing a fraction of the enzyme to the nucleus where it can interact with nuclear substrates such as p53 (correasaez2020updatingdualspecificitytyrosinephosphorylationregulated pages 1-2). Central to the protein is the catalytic kinase domain, which roughly spans residues 222–535. This domain contains the ATP-binding pocket, with key residues such as Lys251 ensuring efficient ATP coordination, and an active site that incorporates conserved residues like Asp348 important for catalytic function. Embedded within the kinase domain is an activation loop that requires autophosphorylation on a conserved tyrosine residue, notably Tyr382, to trigger conformational changes resulting in full kinase activation (carmona2022determinationofnew pages 35-38, correasaez2020updatingdualspecificitytyrosinephosphorylationregulated pages 1-2). In addition, the presence of a DYRK homology box (DH box) contributes to the stability and proper folding of the kinase. High-resolution structural studies and predictive models (such as those generated by AlphaFold) reveal that DYRK2 adopts a typical bilobal kinase fold, with the smaller N-terminal lobe primarily involved in ATP binding and the larger C-terminal lobe housing the substrate-binding region. Unique among its family, DYRK2 also functions as a scaffold within certain E3 ubiquitin ligase complexes, a role that may involve additional interface regions outside of the canonical catalytic domain (correa‑saez2022regulationofposttranslational pages 161-162).
6. Regulation  
   The regulation of DYRK2 is multifaceted and involves several layers of control, including intrinsic post-translational modifications, interactions with regulatory proteins, and transcriptional as well as epigenetic modulation. A key regulatory mechanism is its autophosphorylation on a conserved tyrosine residue within the activation loop, an event that is essential for its conversion from an inactive precursor to an active kinase (correasaez2020updatingdualspecificitytyrosinephosphorylationregulated pages 1-2). In response to genotoxic stress, the ATM kinase phosphorylates DYRK2 on residues Thr33 and Ser369, an event that promotes the stabilization of DYRK2 by reducing its recognition by the ubiquitin ligase MDM2 and facilitating its translocation to the nucleus. Once in the nucleus, DYRK2 is optimally positioned to phosphorylate substrates such as p53 at Ser46 and thereby induce apoptosis (carmona2022determinationofnew pages 35-38, correa‑saez2022regulationofposttranslational pages 161-162). In addition to these phosphorylation events, DYRK2 expression is subject to transcriptional repression; for instance, the transcription factor Kruppel-like factor 4 (KLF4) and promoter methylation mediated by DNMT1 in certain cancers, such as colorectal cancer and chronic myeloid leukemia, serve to downregulate DYRK2 mRNA levels (boni2020thedyrkfamily pages 11-13, correa‑saez2022regulationofposttranslational pages 170-171). Furthermore, microRNAs such as miR-662, miR-208a, and others have been implicated in the post-transcriptional regulation of DYRK2 expression. DYRK2 is also targeted for ubiquitin-dependent proteasomal degradation by E3 ubiquitin ligases such as MDM2 and SIAH2 under different cellular contexts—mechanisms which ensure tight control over its protein abundance (correa‑saez2022regulationofposttranslational pages 162-164, correa‑saez2022regulationofposttranslational pages 170-171). Beyond these modifications, DYRK2 can function as a scaffold for the assembly of the EDVP E3 ligase complex, thereby influencing substrate ubiquitination and degradation independent of its catalytic activity (boni2020thedyrkfamily pages 23-25).
7. Function  
   DYRK2 functions as a serine/threonine protein kinase with broad roles in cellular homeostasis. It is intricately involved in the regulation of the mitotic cell cycle, cellular proliferation, and apoptosis. One of the best-characterized activities of DYRK2 is its phosphorylation of the tumor suppressor protein p53 at Ser46, a modification that is critical for triggering apoptosis in response to DNA damage. This places DYRK2 as a key mediator of the genotoxic stress response downstream of ATM signaling (information section, yan2016lowexpressionof pages 16-17). In addition, DYRK2 phosphorylates NFATC1, thereby preventing its nuclear accumulation and reducing its transcriptional activity; this regulation of NFATC1 contributes to the control of cell survival and possibly immune responses. Furthermore, DYRK2 phosphorylates EIF2B5 at Ser544, which serves as a priming event for subsequent phosphorylation by GSK3β. This sequential phosphorylation event modulates the activity of EIF2B5, thereby influencing protein synthesis and cellular stress responses (information section, correasaez2020updatingdualspecificitytyrosinephosphorylationregulated pages 14-15).  
   Another significant role for DYRK2 is its contribution to the regulation of cytoskeletal organization and neurite outgrowth. By phosphorylating proteins such as CRMP2/DPYSL2 and CRMP4/DPYSL3, DYRK2 primes these substrates for additional phosphorylation by GSK3β, thus participating in signaling pathways that govern cytoskeletal dynamics and neuronal differentiation (information section, boni2020thedyrkfamily pages 15-17). Moreover, DYRK2 inactivates the glycogen synthase enzyme GYS1 through phosphorylation at Ser641 (and possibly at a secondary site), thereby exerting an influence over glycogen synthesis and cellular energy metabolism. In its capacity as a scaffold protein, DYRK2 mediates the formation of the EDVP E3 ligase complex, which is essential for targeting proteins to the proteasome for degradation. This function underscores its broader role in ubiquitin-dependent proteasomal protein turnover, contributing to cell cycle control and maintenance of proteostasis (information section, correa‑saez2022regulationofposttranslational pages 175-176). Collectively, the diverse functions of DYRK2—ranging from cell cycle regulation and apoptotic induction to modulation of cytoskeletal and metabolic pathways—reflect its central involvement in governing cellular proliferation, differentiation, and stress responses (boni2020thedyrkfamily pages 22-23, tandon2021emergingrolesof pages 9-11).
8. Other Comments  
   DYRK2 has been the focus of extensive research due to its dual roles in promoting apoptosis and regulating proteostasis. As such, several small molecule inhibitors have been developed and characterized in preclinical studies. Notable inhibitors include curcumin, harmine, and LDN192960, which have demonstrated efficacy in modulating DYRK2 activity and affecting downstream signaling cascades in cancer cell models (boni2020thedyrkfamily pages 11-13, tandon2021emergingrolesof pages 9-11). These inhibitors serve as valuable tools for dissecting DYRK2 function in vitro and in vivo, although specificity remains a challenge due to the high degree of conservation within the DYRK family.  
   In terms of disease associations, altered expression of DYRK2 has been correlated with various cancers. For example, low DYRK2 expression is associated with poor clinical outcomes in colorectal cancer, lung adenocarcinoma, and certain subtypes of breast cancer, while its overexpression in other contexts such as esophageal and lung adenocarcinomas suggests a complex, context-dependent role (yan2016lowexpressionof pages 14-16, boni2020thedyrkfamily pages 9-11). The ability of DYRK2 to phosphorylate and prime substrates for degradation implicates it in the regulation of oncogenic proteins such as c-Jun, c-Myc, and SNAIL, thereby influencing tumor cell proliferation, invasion, and chemotherapeutic responses. Moreover, its function in the ubiquitin–proteasome system links DYRK2 to broader mechanisms of protein homeostasis that are frequently dysregulated in cancer.  
   Additionally, DYRK2’s involvement in the regulation of neuronal proteins such as NFATC1 and members of the CRMP family suggests potential roles in central nervous system development and neurodegeneration, although these functions remain less well defined compared to its roles in cell cycle and apoptosis. The interplay between DYRK2 and upstream kinases like ATM, as well as its integration into larger regulatory complexes such as the EDVP E3 ligase complex, further underscores its importance as a multifaceted regulator. These multifarious roles have prompted ongoing efforts to develop selective inhibitors and to explore DYRK2 as a therapeutic target in oncology and possibly in other disease contexts such as metabolic disorders and neurodegenerative conditions (information section, tandon2021emergingrolesof pages 17-17).
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