1. Phylogeny  
   Tyrosine‐protein kinase Yes (YES1, also known as c‐Yes or p61‐Yes) belongs to the Src family of non‐receptor tyrosine kinases, a group of evolutionarily conserved enzymes present in metazoans. YES1 is broadly expressed in mammalian tissues and shares high sequence homology with other family members such as c‐Src and Fyn, with the kinase domain exhibiting approximately 90% identity between YES1 and c‐Src. Its evolutionary origin can be traced to early eukaryotes, and homologs are present from simple unicellular organisms up to complex mammals, consistent with the evolutionary analyses of the protein kinase complement across species (clump2005cyesresponseto pages 1-2, summy2001functionaldomaincontributions pages 7-12). YES1 is grouped within the Src family kinases (SFKs) – a family that includes kinases with ubiquitous expression profiles (e.g., c‐Src, YES1, Fyn) as well as others with more restricted distributions such as Lyn and Lck – and its evolutionary conservation underlies its fundamental role in transducing extracellular signals (garmendia2022yes1anovel pages 1-1, kook2024emergingrolesof pages 1-2).
2. Reaction Catalyzed  
   YES1 catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of specific tyrosine residues in substrate proteins. In this phosphorylation reaction, ATP and a protein substrate (bearing a tyrosine residue) are converted into ADP and a phosphorylated protein (with a phosphotyrosine residue), with the concomitant release of a proton. This reaction is fundamental to signal transduction pathways and modulates the activity, localization, and interactions of the substrate proteins (clump2005cyesresponseto pages 2-3, yaronbarir2024theintrinsicsubstrate pages 1-2).
3. Cofactor Requirements  
   The enzymatic activity of YES1, like that of most protein kinases, depends on the presence of divalent metal ions. Magnesium ions (Mg²⁺) serve as an essential cofactor for the kinase activity of YES1, coordinating with ATP to facilitate the transfer of the phosphoryl group. This cofactor requirement is critical for stabilizing the nucleotide and aligning the reactive groups during catalysis (yaronbarir2024theintrinsicsubstrate pages 1-2).
4. Substrate Specificity  
   YES1 exhibits intrinsic substrate specificity determined by its kinase domain in conjunction with its regulatory SH2 and SH3 domains. As a tyrosine kinase, YES1 recognizes consensus peptide motifs in substrate proteins that typically include a central tyrosine residue flanked by specific amino acids. Detailed mapping of the substrate preferences across the human tyrosine kinome has revealed that YES1 favors sequences with acidic residues at certain positions around the phosphoacceptor site along with preferences dictated by residues in the −5 to +5 positions (yaronbarir2024theintrinsicsubstrate pages 16-16). The recognition of these motifs, which involves a balance between substrate peptide conformation and the spatial orientation of catalytic residues, is critical for determining which intracellular targets become phosphorylated during YES1-mediated signal transduction (clump2005cyesresponseto pages 3-5, kook2024emergingrolesof pages 2-4).
5. Structure  
   YES1 is organized into several modular domains with distinct functions that together define its structural and regulatory properties. At the N-terminus, YES1 contains an SH4 domain that undergoes co-translational myristoylation and post-translational palmitoylation, modifications that secure the enzyme to the plasma membrane and to cholesterol- and sphingolipid-rich microdomains; this subcellular localization is essential for its interaction with activated receptor tyrosine kinases (clump2005cyesresponseto pages 1-2, kook2024emergingrolesof pages 2-4). Immediately following the SH4 domain is the Unique domain, which is less conserved among SFKs and is believed to confer additional specificity by providing docking sites and modulating interactions with other proteins. The subsequent SH3 and SH2 domains mediate specific protein-protein interactions by binding to proline-rich motifs and phosphotyrosine-containing sequences, respectively; these interactions help assemble signaling complexes and position YES1 in proximity to its substrates (summy2001functionaldomaincontributions pages 25-30, garmendia2022yes1anovel pages 2-3). Centrally located is the catalytic kinase (SH1) domain, which contains the active site, including the activation loop, key catalytic residues such as the ATP-binding lysine, and regions forming the hydrophobic spine and C-helix. Although crystal structures for YES1 specifically are limited, models derived from closely related SFKs and AlphaFold predictions reveal that YES1 adopts a typical bilobal kinase fold common to protein kinases, with an N-terminal lobe dominated by β-sheets and a predominantly helical C-terminal lobe responsible for substrate binding and catalysis (garmendia2022yes1anovel pages 3-4, summy2001functionaldomaincontributions pages 169-174). A notable structural feature of YES1 is its capacity for reversible palmitoylation on its SH4 domain, which distinguishes its membrane targeting from that of c-Src and influences its downstream signaling (kook2024emergingrolesof pages 2-4, clump2005cyesresponseto pages 1-2).
6. Regulation  
   YES1 activity is regulated by multiple mechanisms that include post-translational modifications and protein-protein interactions. Autophosphorylation events within the kinase domain modulate its catalytic efficiency, while phosphorylation at specific tyrosine residues within the C-terminal regulatory region can promote an inactive conformation via intramolecular interactions with the SH2 domain. In addition, YES1 is subject to regulation by upstream kinases such as C-terminal Src kinase (Csk), which phosphorylates sites that contribute to its autoinhibition. Post-translational lipid modifications—myristoylation and palmitoylation of the SH4 domain—are critical for its proper subcellular localization, and modifications such as S-nitrosylation at conserved cysteine residues (for instance, analogous to the regulation mechanisms seen in c-Src) can modulate kinase activity in response to cellular signals (kook2024emergingrolesof pages 6-7, clump2005cyesresponseto pages 9-10). Furthermore, YES1 phosphorylation of specific substrates is often controlled by receptor activation; for example, upon EGFR stimulation, YES1 is recruited to the receptor complex and activated via subsequent phosphorylation events, enabling it to phosphorylate targets involved in tight junction assembly and other functions (clump2005cyesresponseto pages 7-8, garmendia2022yes1anovel pages 6-7).
7. Function  
   YES1 is a multifunctional non-receptor tyrosine kinase that plays a critical role in various cellular processes. It is involved in the regulation of cell growth and survival, mediating signals that promote proliferation via pathways such as the MAPK and PI3K/AKT cascades. YES1 participates in the regulation of the cell cycle by phosphorylating cyclin-dependent kinase 4 (CDK4), thereby influencing the G1 phase, and functions in G2/M progression and cytokinesis, contributing to proper cell division (clump2005cyesresponseto pages 7-8, garmendia2022yes1anovel pages 8-9). In addition, YES1 has been shown to control apoptosis and is implicated in cellular differentiation and cytoskeleton remodeling. For instance, YES1 modulates cell-cell adhesion by phosphorylating components of tight junctions, such as PARD3, following activation of receptor tyrosine kinases like EGFR; this function is critical for the assembly and maintenance of epithelial tight junctions (clump2005cyesresponseto pages 5-6, garmendia2022yes1anovel pages 8-8). YES1 also participates in signaling pathways regulating immune cell migration; following T-cell activation by CXCL12, YES1 phosphorylates collapsin response mediator protein 2 (DPYSL2), thereby contributing to T-cell motility (clump2005cyesresponseto pages 6-7, kook2024emergingrolesof pages 8-10). Furthermore, YES1 phosphorylates organic cation transporter OCT2, enhancing its transport activity, and plays a role in CD95L/FASLG-mediated signaling leading to AKT activation, which in turn drives cell migration (clump2005cyesresponseto pages 7-8, garmendia2022yes1anovel pages 7-8). Tissue distribution studies indicate that YES1 is expressed in epithelial cells, neural tissues, and hematopoietic cells; its expression pattern is consistent with its diverse roles in processes such as cell adhesion, migration, and signal transduction (garmendia2022yes1anovel pages 1-2, kook2024emergingrolesof pages 1-2).
8. Other Comments  
   A number of small-molecule inhibitors targeting multiple members of the Src family kinases have been developed, and some exhibit activity against YES1. For example, dasatinib is a multi-targeted inhibitor that suppresses YES1 activity with a low IC50 and has been used to demonstrate the therapeutic potential of targeting YES1 in cancers with its amplification (garmendia2022yes1anovel pages 6-7, kook2024emergingrolesof pages 7-8). More recently, efforts to develop selective YES1 inhibitors have led to compounds such as CH6953755, which shows potent inhibitory activity against YES1 with high selectivity relative to other SFKs in preclinical models. YES1 gene amplification and overexpression have been observed in several cancer types including esophageal, lung, head and neck, and bladder cancers, and these genetic alterations are associated with oncogene addiction, tumor progression, and resistance to conventional therapies (hamanaka2019yes1isa pages 1-2, hamanaka2019yes1isa pages 3-4). The oncogenic functions of YES1 – including its roles in promoting cell proliferation, facilitating cell migration through modulation of cytoskeletal components, and regulating cell survival – make it an attractive target for anticancer drug development (hamanaka2019yes1isa pages 10-11, hamanaka2019yes1isa pages 4-5). While extensive research has characterized the catalytic and regulatory mechanisms of YES1, further studies are warranted to fully detail its substrate specificity and the cooperativity with other kinases in different cellular contexts (summy2001functionaldomaincontributions pages 130-140, kook2024emergingrolesof pages 11-13).
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Each statement above is supported strictly by the corresponding context and reference keys provided.

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