1. Phylogeny  
   Tyrosine‐protein kinase Fyn is a member of the Src family kinases (SFKs), a group of non‐receptor tyrosine kinases that are conserved across metazoans and are present in all mammalian species (huculeci2016dynamicallycoupledresidues pages 1-2). Fyn shares strong sequence and structural similarity with other Src‐related kinases such as Src, Yes, and Fgr, and is phylogenetically distinct from the Hck‐related subgroup that includes Lyn, Hck, and Lck (register2014sh2catalyticdomainlinker pages 1-3). In addition, the existence of alternatively spliced isoforms––notably Fyn1 and Fyn2 which differ in the length and sequence of the SH2–catalytic domain linker––further supports its evolutionary diversification within the SFK family (brignatz2009alternativesplicingmodulates pages 1-2, register2014sh2catalyticdomainlinker pages 1-3).
2. Reaction Catalyzed  
   Fyn catalyzes a phosphorylation reaction in which a phosphate group is transferred from ATP to a tyrosine residue on a protein substrate, resulting in the formation of ADP, a phosphorylated protein, and a proton (sakkiah2017overviewofthe pages 2-3).
3. Cofactor Requirements  
   The kinase activity of Fyn requires divalent metal ions as cofactors, with Mg²⁺ being essential for efficient ATP binding and phosphate transfer during the phosphorylation reaction (sakkiah2017overviewofthe pages 2-3).
4. Substrate Specificity  
   Fyn phosphorylates protein substrates on tyrosine residues and exhibits substrate specificity that is determined by the presence of specific amino acid motifs surrounding the target tyrosine. For example, Fyn phosphorylates tau protein at Tyr18 and beta‐catenin as well as delta‐catenin, which participate in pathways related to cytoskeletal dynamics and cell adhesion (demuro2022arn25068aversatile pages 1-3, bhaskar2005diseaserelatedmodificationsin pages 1-1). In addition, substrates such as the phosphatidylinositol 3-kinase enhancer (PIKE-A) are phosphorylated by Fyn to promote cell survival by inhibiting apoptotic cleavage (tang2007srcfamilytyrosinekinase pages 9-10). Although a precise consensus substrate motif for Fyn is not explicitly defined in the available context, the kinase’s activity on substrates with phosphotyrosine motifs indicates it recognizes local sequence elements that facilitate SH2 domain-mediated interactions (brignatz2009alternativesplicingmodulates pages 1-2).
5. Structure  
   Fyn is organized into several distinct domains that together facilitate its catalytic and regulatory functions. The protein contains an N-terminal SH4 domain that undergoes fatty acylation (myristoylation and palmitoylation) for membrane targeting, which is critical for its proper localization within specific plasma membrane microdomains such as lipid rafts (ouyang2018sensitivefretbiosensor pages 1-4, jelic2007homologymodelingof pages 1-2). Immediately following the SH4 domain, the SH3 domain mediates protein–protein interactions through binding to proline-rich sequences, while the SH2 domain recognizes phosphotyrosine-containing motifs; both domains play key roles in the autoinhibition and activation of the kinase (register2014sh2catalyticdomainlinker pages 1-3, huculeci2016dynamicallycoupledresidues pages 1-2). The catalytic domain (commonly referred to as SH1) is centrally located and contains the activation loop, an αC helix that contributes to the formation of a hydrophobic spine, and a conserved ATP-binding pocket in which key residues such as those in the hinge region (e.g., Glu343 and Met345) participate in ligand interactions (demuro2022arn25068aversatile pages 1-3, jelic2007homologymodelingof pages 7-7). Fyn also harbors a C-terminal tail that includes regulatory tyrosine residues, which when phosphorylated by Csk contribute to maintaining the protein in an autoinhibited conformation (andoniou2000thecblprotooncogene pages 1-2, register2014sh2catalyticdomainlinker pages 1-3). Structural studies based on homology modelling and X-ray crystallography have provided insights into these domain interactions and the dynamic allosteric coupling between the SH2, SH3, and catalytic domains (jelic2007homologymodelingof pages 2-3, register2014sh2catalyticdomainlinker pages 3-4).
6. Regulation  
   Fyn activity is intricately regulated by multiple post-translational modifications and intermolecular interactions. Autoinhibition of Fyn is achieved through intramolecular interactions in which the SH2 domain binds to a phosphorylated tyrosine residue in the C-terminal tail, and the SH3 domain interacts with a proline-rich region in the linker domain; these interactions restrain the catalytic domain and maintain Fyn in a low activity state (register2014sh2catalyticdomainlinker pages 1-3, brignatz2009alternativesplicingmodulates pages 3-5). Activation of Fyn involves dephosphorylation of the inhibitory C-terminal tyrosine and subsequent phosphorylation of the activation loop tyrosine by autophosphorylation, which increases catalytic activity (demuro2022arn25068aversatile pages 1-3, weir2016novelautophosphorylationsites pages 1-3). Additional regulation is provided by alternative splicing that produces isoforms (Fyn1 and Fyn2) with different regulatory properties due to variations in their SH2–catalytic domain linker, thereby altering allosteric coupling and substrate binding potential (brignatz2009alternativesplicingmodulates pages 7-9, register2014sh2catalyticdomainlinker pages 3-4). Negative regulation is also exerted through protein–protein interactions; for instance, the c-Cbl proto-oncogene product binds to Fyn and promotes its ubiquitination and subsequent proteasomal degradation (andoniou2000thecblprotooncogene pages 1-2, andoniou2000thecblprotooncogene pages 15-16). Moreover, novel autophosphorylation sites within the SH2 domain, such as Tyr185, Tyr213, and Tyr214, modulate both kinase activity and the phosphotyrosine-binding capacity of the SH2 domain, thus impacting downstream signaling interactions (weir2016novelautophosphorylationsites pages 4-6, weir2016novelautophosphorylationsites pages 9-13). Fyn is further regulated by upstream kinases and phosphatases that modulate its phosphorylation status in response to extracellular signals; for example, C-terminal Src kinase (Csk) phosphorylates the inhibitory tyrosine, while protein kinase A (PKA) has been reported to activate Fyn indirectly by facilitating its association with focal adhesion kinase (PTK2/FAK1) (demuro2022arn25068aversatile pages 1-3, andoniou2000thecblprotooncogene pages 12-13).
7. Function  
   Fyn plays multiple roles in diverse biological processes and is expressed in a wide variety of tissues, with particularly high levels in neural and immune cells. In neuronal cells, Fyn regulates cytoskeletal remodeling by phosphorylating microtubule-associated proteins such as MAPT (tau) and MAP2, thereby influencing axon guidance and synaptic plasticity; it also participates in reelin signaling by mediating the phosphorylation of DAB1 (demuro2022arn25068aversatile pages 1-3, brignatz2009alternativesplicingmodulates pages 1-2). In the immune system, Fyn is a critical mediator of T-cell receptor (TCR) signaling, where it phosphorylates substrates such as VAV1 and components of the CD28 pathway, thereby promoting T-cell differentiation and proliferation; Fyn also plays a role in mast cell and natural killer (NK) cell signaling (marotta2022roleoffyn pages 1-1, mkaddem2017lynandfyn pages 1-2). Fyn is involved in integrin-mediated signaling processes that regulate cell adhesion and motility through the phosphorylation of proteins such as CTNNB1 (beta-catenin) and CTNND1 (delta-catenin), which contribute to focal adhesion turnover and cytoskeletal reorganization (demuro2022arn25068aversatile pages 1-3, marotta2022roleoffyn pages 1-3). Additionally, Fyn enhances cell survival by phosphorylating proteins such as AGAP2/PIKE-A, thus preventing apoptotic cleavage and promoting antiapoptotic signaling via the Akt pathway (tang2007srcfamilytyrosinekinase pages 9-10, saminathan2021fynkinasemediatedpkcδ pages 1-2). In the kidney, Fyn is important for maintaining the integrity of the glomerular filter by phosphorylating essential components of the slit diaphragm, including NPHS1 and TRPC6, and in neural tissues, Fyn modulates signal transduction processes that contribute to learning and memory (demuro2022arn25068aversatile pages 1-3, marotta2022roleoffyn pages 15-16).
8. Other Comments  
   Several small molecule inhibitors that target the ATP‐binding pocket of Fyn have been developed and evaluated for their therapeutic potential. Among these, non-selective agents such as dasatinib and saracatinib have been repurposed for clinical investigation in neurodegenerative disorders and cancers, although their lack of specificity across the Src family limits their clinical utility (marotta2022roleoffyn pages 4-6, comba2020fyntyrosinekinase pages 11-12). Additionally, natural compounds such as rosmarinic acid have been identified as Fyn inhibitors through in silico docking and enzymatic assays, acting by a mixed-type inhibition mechanism that may involve binding to an allosteric site in addition to the canonical ATP-binding pocket (jelic2007homologymodelingof pages 7-8, marotta2022roleoffyn pages 7-9). Disease associations of Fyn include its implication in Alzheimer’s disease due to its role in tau phosphorylation and in various cancers, where its deregulated activity contributes to aberrant cell survival and migration (demuro2022arn25068aversatile pages 1-3, mkaddem2017lynandfyn pages 1-2). Moreover, genetic and biochemical studies have linked Fyn to immune dysfunctions, including defective T-cell receptor signaling and autoimmunity, as well as to neurodevelopmental processes that may impact central nervous system disorders (marotta2022roleoffyn pages 1-1, ouyang2018sensitivefretbiosensor pages 17-20).
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