1. Phylogeny  
   Tyrosine‐protein kinase Fyn is a member of the Src family kinases (SFKs), a sub‐group of non‐receptor tyrosine kinases that are conserved across metazoans. Fyn is ubiquitously expressed in mammalian species and has identifiable orthologs across vertebrates that share a common domain organization and regulatory mechanism with other SFKs such as c‐Src, Yes, and Lyn (roskoski2004srcprotein–tyrosinekinase pages 1-2, ingley2008srcfamilykinases pages 1-2). Fyn is placed within the evolutionary branch that emerged early in eukaryotic evolution, and phylogenetic studies based on the kinase complement of the human genome have demonstrated that Fyn and its related kinases trace their origin to a common ancestral kinase predating the divergence between yeast and man. Its evolutionary conservation is underscored by the characteristic modular structure comprising an N‐terminal SH4 domain, a unique region, SH3 and SH2 domains, followed by the catalytic kinase domain and a C‐terminal regulatory tail (marotta2022roleoffyn pages 1-1, roskoski2004srcprotein–tyrosinekinase pages 1-2).
2. Reaction Catalyzed  
   Tyrosine‐protein kinase Fyn catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of tyrosine residues present on substrate proteins. The generalized chemical reaction is:  
     ATP + [protein – tyrosine] → ADP + [protein – phosphotyrosine] + H⁺  
   This phosphorylation reaction is fundamental to cell signaling pathways and is involved in modulating the activity, interaction, and subcellular location of numerous target proteins (jelic2007homologymodelingof pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of Fyn kinase requires the presence of divalent metal ions; specifically, Mg²⁺ is essential for forming a stable complex with ATP within the active site. This cofactor facilitates proper positioning of the ATP molecule and is necessary for efficient phosphoryl transfer to substrates (cowanjacob2006structuralbiologyof pages 1-2).
4. Substrate Specificity  
   The intrinsic substrate specificity of Fyn, as a member of the human tyrosine kinome, has been profiled using combinatorial peptide arrays and high‐throughput methods. Fyn shows a preference for target tyrosine residues embedded within peptide motifs that typically include a hydrophobic residue preceding the phosphoacceptor tyrosine and a polar or acidic residue immediately following it. This selectivity is consistent with the broader substrate preferences of Src family kinases, which rely on interactions mediated by their SH2 and SH3 domains to direct phosphorylation events (yaronbarir2024theintrinsicsubstrate pages 1-2).
5. Structure  
   Fyn kinase is organized into several conserved domains that are crucial for its catalytic function and regulatory interactions. At the very N‐terminus, the SH4 domain, which undergoes myristoylation and in some cases palmitoylation, mediates membrane attachment and subcellular localization (kinoshita2006structureofhuman pages 1-2, matrone2020fyntyrosinekinase pages 3-6). Following the SH4 domain is a unique region that is less conserved; this region can help determine specific protein–protein interactions. Next, the SH3 domain, typically around 50 amino acids, recognizes proline‐rich motifs in interacting partners, thereby contributing to both intramolecular regulation and substrate binding (roskoski2004srcprotein–tyrosinekinase pages 1-2, ingley2008srcfamilykinases pages 1-2). The subsequent SH2 domain, approximately 100 amino acids in length, binds to phosphotyrosine motifs present on target proteins with specificity dictated by surrounding amino acid sequences. Centrally located is the catalytic or kinase domain (SH1 domain), which adopts a bilobed structure; the smaller N‐terminal lobe is involved in ATP binding, whereas the larger C‐terminal lobe confers substrate specificity. Key structural features of this catalytic domain include the activation loop, a conserved C‐helix, and the hydrophobic spine that stabilizes the active conformation. A short C‐terminal tail contains a regulatory tyrosine residue that, when phosphorylated, binds intramolecularly to the SH2 domain, leading to the autoinhibited conformation of Fyn (jelic2007homologymodelingof pages 2-3, matrone2020fyntyrosinekinase pages 8-10, roskoski2004srcprotein–tyrosinekinase pages 1-2). Several high‐resolution crystal structures and homology models, based on templates with 70–80% sequence identity such as c‐Src, have been used to delineate these structural features in Fyn (jelic2007homologymodelingof pages 8-9).
6. Regulation  
   Fyn regulation occurs via multiple post‐translational modifications and protein–protein interactions. A primary regulatory mechanism is phosphorylation. In its inactive state, Fyn is phosphorylated at a conserved C‐terminal tyrosine (for example, Tyr531 in FynB isoform), which facilitates an intramolecular interaction with the SH2 domain that maintains the kinase in a closed conformation (matrone2020fyntyrosinekinase pages 1-3, taleski2020thederegulationof pages 28-31). Activation of Fyn involves dephosphorylation of this inhibitory site coupled with autophosphorylation of a tyrosine residue in the activation loop (Tyr420 in the FynB isoform) that stabilizes the active configuration of the catalytic domain (taleski2020thederegulationof pages 31-34, crosby2003physicalandfunctional pages 4-5). In addition to phosphorylation events, Fyn activity is modulated by lipid modifications such as myristoylation and palmitoylation at the SH4 domain, which are required for its association with the plasma membrane and lipid rafts (kinoshita2006structureofhuman pages 1-2, matrone2020fyntyrosinekinase pages 3-6). Fyn also interacts with regulatory proteins such as C-terminal Src kinase (CSK); CSK phosphorylates the inhibitory C-terminal tyrosine and thereby contributes to negative feedback regulation (parravicini2002fynkinaseinitiates pages 5-5, taleski2020thederegulationof pages 77-80).
7. Function  
   Fyn kinase plays diverse roles in numerous biological processes. It is involved in the regulation of cell growth, survival, and motility, and it mediates signal transduction downstream of multiple receptors. In the context of cell adhesion and integrin-mediated signaling, Fyn phosphorylates proteins such as beta-catenin (CTNNB1) and delta-catenin (CTNND1), thereby influencing cytoskeletal remodeling and cellular motility (jelic2007homologymodelingof pages 1-2). Fyn also regulates actin dynamics and microtubule stability by phosphorylating actin regulatory proteins and microtubule-associated proteins such as MAP2 and MAPT (matrone2020fyntyrosinekinase pages 10-12). In immune cells, Fyn participates in T-cell receptor (TCR) signaling by phosphorylating key substrates including PTK2B/PYK2 and PAG1; these phosphorylation events modulate both positive signaling cascades that promote T-cell differentiation and negative feedback loops that limit TCR signaling (jin2017anessentialrole pages 1-2, parravicini2002fynkinaseinitiates pages 5-5). In mast cells, Fyn phosphorylates adaptor proteins such as CLNK following immunoglobulin epsilon receptor activation, contributing to degranulation responses (parravicini2002fynkinaseinitiates pages 5-5). In neuronal cells, Fyn is critical for processes including axon guidance, neural migration, and synaptic plasticity; it phosphorylates substrates like DPYSL2 and ARHGAP32 and interacts with factors implicated in reelin signaling (jin2017anessentialrole pages 13-14, matrone2020fyntyrosinekinase pages 12-14). Furthermore, Fyn is involved in the phosphorylation of proteins at the glomerular slit diaphragm, implicating it in the regulation of kidney filtration processes (jelic2007homologymodelingof pages 1-2). Collectively, these diverse roles underscore Fyn kinase as an integrator of signaling pathways that coordinate cellular adhesion, cytoskeletal architecture, immune cell activation, and neural function (matrone2020fyntyrosinekinase pages 1-3, taleski2020thederegulationof pages 31-34).
8. Other Comments  
   Several small-molecule inhibitors have been identified and developed to target Fyn kinase activity. Among these, natural compounds such as rosmarinic acid have been characterized as non-ATP competitive inhibitors that bind to alternative pockets on the kinase surface, while broad-spectrum inhibitors like staurosporine exhibit ATP-competitive inhibition (jelic2007homologymodelingof pages 9-10, marotta2022roleoffyn pages 7-9). In addition, drugs such as dasatinib and saracatinib, initially developed for oncological indications, have been repurposed due to their activity against Fyn in preclinical models of neurodegeneration and immune disorders (marotta2022roleoffyn pages 4-6, taleski2020thederegulationof pages 77-80). Dysregulated Fyn activity has been associated with various pathological conditions including cancer, autoimmune disorders, and neurodegenerative diseases such as Alzheimer’s disease, where aberrant Fyn signaling contributes to synaptic dysfunction and tau hyperphosphorylation (taleski2020thederegulationof pages 80-83, marotta2022roleoffyn pages 11-12). The promiscuity associated with the conserved ATP-binding pocket among Src family kinases has presented challenges in the development of highly selective Fyn inhibitors; therefore, ongoing research focuses on developing compounds that exploit subtle structural differences or alternative binding sites to maximize selectivity (passannanti2021applicationofcomputational pages 120-122, roskoski2004srcprotein–tyrosinekinase pages 2-3).
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