1. Phylogeny  
   Tyrosine‐protein kinase Fgr is a member of the Src family kinases (SFKs), a well‐defined subgroup of non‐receptor tyrosine kinases conserved from invertebrates to vertebrates. Fgr shares extensive sequence and structural homology with classical SFK members such as Src, Fyn, Yes, Lyn, Hck, Blk, and Lck. Within the mammalian kinome, Fgr is predominantly expressed in hematopoietic cells and is phylogenetically classified among kinases that are critical regulators of immune responses. Its evolutionary conservation is evident across multiple species – orthologous sequences have been identified in common model organisms, which supports the notion that Fgr evolved from an ancestral SFK present in the Last Eukaryotic Common Ancestor (LECA) (santos2016paralogspecificpatternsof pages 1-1, sekar2023kinaceaweb pages 14-17, shen2018thesrcfamily pages 10-11, patel2019srcfamilykinasesimpact pages 68-73).
2. Reaction Catalyzed  
   Fgr catalyzes the transfer of the γ‐phosphate group from adenosine triphosphate (ATP) to tyrosine residues within target protein substrates. In this reaction mechanism, ATP is converted into adenosine diphosphate (ADP) while a hydroxyl group (–OH) on a substrate tyrosine performs a nucleophilic attack on the γ‐phosphate of ATP, forming a phosphotyrosine residue. This phosphorylation event is a key post‐translational modification that regulates protein activity, subcellular localization, and interactions, thereby triggering downstream signaling cascades critical for immune cell activation and cytoskeletal reorganization (eshaq2024nonreceptortyrosinekinases pages 2-4, shen2018thesrcfamily pages 8-9, weir2018selectiveinhibitionof pages 1-4).
3. Cofactor Requirements  
   The catalytic activity of Fgr is dependent upon divalent metal ions, with Mg²⁺ serving as the primary cofactor. Mg²⁺ is essential as it coordinates with ATP in the kinase active site, stabilizing the molecule and neutralizing the negative charges on the phosphate groups, thereby facilitating efficient phosphotransfer. Although in some kinase systems substitution with Mn²⁺ has been observed under specific conditions, the biochemical characterization of SFKs such as Fgr overwhelmingly indicates that optimal Fgr activity requires Mg²⁺. This cofactor dependency aligns with the general property of tyrosine kinases, where coordination of ATP by a divalent metal ion is critical for catalysis (du2022atpsiteinhibitorsinduce pages 1-3, patel2019srcfamilykinasesimpact pages 68-73, eshaq2024nonreceptortyrosinekinases pages 2-4).
4. Substrate Specificity  
   Fgr phosphorylates an array of substrates that are centrally involved in immune receptor signaling and cytoskeletal regulation. A key substrate is Spleen tyrosine kinase (SYK), phosphorylation of which by Fgr in vitro has been shown to promote activation of downstream signaling involving AKT1 and the MAP kinase cascade. In mast cells Fgr phosphorylates phospholipase D2 (PLD2), leading to the production of critical lipid mediators such as lysophosphatidic acid and diacylglycerol. Other noteworthy substrates include the FAS ligand (FASLG), where phosphorylation affects its ubiquitination and subsequent internalization, as well as ABL1, and adaptor proteins such as CBL and cortactin (CTTN). Furthermore, Fgr phosphorylates key components of integrin-mediated signaling pathways including the PI3K regulatory subunit (PIK3R1) and the focal adhesion kinases PTK2 (FAK1) and PTK2B (PYK2). VAV2 and HCLS1 are additional substrates, with the latter being phosphorylated only when pre-modified by SYK. Although there is no single consensus phosphorylation motif defined for Fgr analogous to motifs recognized by serine/threonine kinases, substrate specificity is mediated in part by protein–protein interactions through its SH2 and SH3 domains. These domains either recognize phosphotyrosine residues or bind proline-rich sequences that are present on target proteins, thereby directing Fgr to its appropriate substrates (abe2019gainoffunctionmutationsin pages 1-1, ciapala2017thesrcfamilykinase pages 90-93, patel2019srcfamilykinasesimpact pages 73-77, du2022atpsiteinhibitorsinduce pages 3-5, patel2019srcfamilykinasesimpacta pages 73-77).
5. Structure  
   Fgr exhibits the canonical domain organization that is characteristic of Src family kinases. At the N-terminus, it possesses an SH4 domain responsible for membrane anchorage via lipid modifications such as myristoylation and possibly palmitoylation. This membrane-targeting process is crucial for positioning Fgr in close proximity to its receptor substrates (gormal2024locationlocationlocation pages 18-19). Next, Fgr contains a Unique region whose sequence diverges considerably from its SFK relatives; this region may contribute to differential protein–protein interactions, although its precise function is less well characterized. Continuing along the polypeptide chain is the SH3 domain, a small module that recognizes proline-rich motifs in partner proteins and participates in both intramolecular and intermolecular interactions that can influence the kinase’s conformation. Adjacent to the SH3 domain is the SH2 domain, which specifically binds phosphotyrosine-containing sequences; this domain plays a central role in both substrate recognition and the autoregulatory mechanism by interacting with a phosphorylated tyrosine in the C-terminal tail in many SFKs. Central to the structure is the catalytic kinase domain (SH1), a bilobed structure composed of N- and C-terminal lobes that form the ATP binding site and the substrate binding cleft. Within this kinase domain resides an activation loop, which in Fgr is noted for having unique features, such as an atypical amino acid substitution near the critical autophosphorylation site analogous to Tyr416 in Src. This distinct activation loop is associated with a higher basal kinase activity compared to other SFKs and is implicated in Fgr’s potent transforming ability (shen2018thesrcfamily pages 10-11, ciapala2017thesrcfamilykinase pages 26-30, du2022atpsiteinhibitorsinduce pages 3-5). In addition, Fgr contains a short C-terminal tail harboring a regulatory tyrosine residue; in many Src kinases, phosphorylation of this residue (equivalent to Tyr527 in Src) mediates autoinhibition by engaging the SH2 domain. However, in Fgr the presence of this phosphorylated tail does not result in classical inhibitory interactions, contributing further to its relatively high basal activity. Although full-length high-resolution crystallographic structures of Fgr remain limited, hydrogen–deuterium exchange mass spectrometry (HDX-MS) studies confirm that its overall fold is similar to other SFKs, while significant displacements in the SH3–SH2 regulatory module imply a divergence in autoinhibitory control and substrate accessibility (bagnato2020nuclearfunctionsof pages 1-3, shen2018thesrcfamily pages 5-6, passannanti2021applicationofcomputational pages 14-17, kinoshitakikuta2022characterizationofphosphorylation pages 10-11).
6. Regulation  
   The regulatory mechanisms governing Fgr are multifactorial and involve phosphorylation dynamics, conformational rearrangements, and protein–protein interactions mediated by its modular domains. Activation of Fgr critically depends on autophosphorylation of a tyrosine residue within the activation loop (analogous to Tyr416 in Src), which facilitates a shift toward an active kinase conformation. In many Src family kinases, a conserved tyrosine residue in the C-terminal tail (comparable to Tyr527 in Src) is phosphorylated by C-terminal Src kinase (CSK), triggering an intramolecular interaction with the SH2 domain and enforcing an inactive “closed” conformation. In contrast, although Fgr is phosphorylated on its C-terminal tail, several studies have demonstrated that this phosphorylation does not effectively suppress its kinase activity. Fgr’s activation loop possesses a unique amino acid motif – including a proline residue at the +2 position relative to the activation loop tyrosine – that contributes to its elevated basal activity and attenuates classical SH3-SH2-mediated autoinhibition. Mutational studies have shown that substituting Fgr’s activation loop with that of Src reduces both kinase activity and transformative ability, underscoring the importance of this region in dictating its regulatory behavior (shen2018thesrcfamily pages 1-2, shen2018thesrcfamily pages 10-11, shu2025constitutiveactivationof pages 1-2).  
   Furthermore, regulatory inputs in Fgr are modulated by intermolecular interactions with adaptor proteins and may be influenced by the binding of ATP-site inhibitors. For instance, small molecules such as A-419259 and TL02-59 have been reported to induce allosteric shifts in Fgr’s conformation, resulting in altered exposure of the SH3 and SH2 domains and influencing overall kinase activity (du2022atpsiteinhibitorsinduce pages 11-13, weir2018selectiveinhibitionof pages 8-11). These studies collectively suggest that Fgr functions with a regulatory scheme that is partly independent of the canonical SH3/SH2 autoinhibitory interaction prevalent in other Src family members, enabling sustained activity that might underpin its oncogenic potential in certain hematopoietic contexts.
7. Function  
   Fgr is intimately involved in the regulation of immune signaling. It transmits signals from cell surface receptors lacking intrinsic kinase activity – notably receptors binding the Fc portion of immunoglobulins such as MS4A2/FCER1B, FCGR2A, and FCGR2B, as well as integrins ITGB1 and ITGB2. In immune cells including neutrophils, monocytes, macrophages, and mast cells, Fgr regulates critical processes such as cytoskeletal reorganization, cell adhesion, migration, spreading, and phagocytosis. These functions are essential for an effective immune response and include both activating and inhibitory roles.  
   In mast cells, for example, upon antigen stimulation, Fgr phosphorylates PLD2, which catalyzes the production of lipid signaling molecules (lysophosphatidic acid and diacylglycerol) that drive degranulation and the release of inflammatory cytokines, thereby playing a role in IgE-mediated anaphylaxis. In parallel, Fgr phosphorylates SYK – a key kinase in immune receptor signaling – to initiate downstream activation of AKT1 and MAP kinase pathways, which are essential for cell migration and survival. Beyond its role in promoting activation, Fgr has been shown to act as a negative regulator in specific contexts, as it modulates ITGB2 signaling and phagocytic activity in monocytes. In contrast, in neutrophils and macrophages, Fgr is required for proper integrin-mediated signaling essential for normal adhesion and spreading (abe2019gainoffunctionmutationsin pages 1-1, du2022atpsiteinhibitorsinduce pages 6-8, patel2019srcfamilykinasesimpact pages 73-77).  
   Additionally, Fgr phosphorylates FASLG, thereby influencing its ubiquitination and subsequent internalization, which may affect the balance between activation and apoptosis in immune cells. The kinase also contributes to focal adhesion dynamics through the phosphorylation of ABL1, CBL, cortactin (CTTN), and focal adhesion kinases such as PTK2/FAK1 and PTK2B/PYK2, while also engaging with VAV2 and HCLS1 (the latter being phosphorylated only when previously modified by SYK). Moreover, in partnership with the adaptor protein CLNK, Fgr serves as a negative regulator of natural killer cell activating receptors and suppresses interferon‐γ production, thereby fine-tuning the immune response (ciapala2017thesrcfamilykinase pages 90-93, shen2018thesrcfamily pages 8-9, eshaq2024nonreceptortyrosinekinases pages 20-21).  
   Clinically, aberrant expression and constitutive activation of Fgr have been implicated in acute myeloid leukemia (AML), where overexpression contributes to cytokine-independent proliferation and survival of leukemic cells. The transforming potential of Fgr has been demonstrated in fibroblast transformation assays, and RNA interference studies in primary AML cells have underscored its importance in leukemic proliferation, making it a promising target for therapeutic intervention (shen2018thesrcfamily pages 1-2, patel2019srcfamilykinasesimpact pages 73-77, shu2025constitutiveactivationof pages 1-2).
8. Other Comments  
   Selective inhibition of Fgr is a current focus of research, particularly given its dual role in both immune activation and suppression, and its emerging importance in hematopoietic malignancies such as AML. ATP-site inhibitors like A-419259 and TL02-59 have been shown in preclinical models to inhibit Fgr autophosphorylation and downstream signaling pathways that modulate cell adhesion, migration, and cytokine production (du2022atpsiteinhibitorsinduce pages 14-18, weir2018selectiveinhibitionof pages 8-11). Such compounds not only suppress the proliferation of leukemic cells but also alter immune cell functions, highlighting the therapeutic potential of targeting Fgr specifically.  
   In addition, the unique regulatory features of Fgr – particularly its relative insensitivity to classical SH3–SH2 mediated autoinhibition due to a distinctive activation loop – make it an attractive candidate for the development of inhibitors that can selectively target Fgr without significantly affecting other SFK members. This selectivity is important to limit off-target effects, given the broad expression and critical physiological roles of other Src family kinases in non-hematopoietic tissues.  
   Current research efforts are focused on further elucidating the three-dimensional structure of full-length Fgr, mapping its complete substrate repertoire, and defining the contributions of post-translational modifications to its function in different cellular contexts. New technological approaches, such as deep learning-coupled proximity assays and advanced HDX-MS techniques, are being employed to capture the dynamic regulatory conformations of Fgr, which in turn may inform the design of next-generation, highly selective inhibitors (jha2025deeplearningcoupledproximity pages 24-26, amatya2019dynamicregulatoryfeatures pages 1-3, passannanti2021applicationofcomputational pages 14-17).  
   Furthermore, Fgr’s contrasting roles – serving as a positive regulator in integrin-mediated signaling in neutrophils and macrophages while acting as a negative regulator in monocytes – underscore the complexity of its function in immune homeostasis and inflammation. These context-dependent activities provide valuable insights into how modulation of Fgr activity can be leveraged to treat diverse pathologies ranging from inflammatory disorders to hematologic cancers (eshaq2024nonreceptortyrosinekinases pages 20-21, bagnato2020nuclearfunctionsof pages 1-3, shen2018thesrcfamily pages 10-11).  
   There is also considerable interest in using Fgr expression levels as a biomarker for immune cell lineage specification and for predicting responsiveness to targeted kinase inhibition in AML. As further studies define the molecular determinants of its substrate interactions and inhibitor binding, it is anticipated that more potent and selective Fgr inhibitors will enter clinical evaluation, potentially in combination with other therapies aiming at dysregulated signaling networks in leukemogenesis (du2022atpsiteinhibitorsinduce pages 14-18, passannanti2021applicationofcomputational pages 17-21).
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