1. Phylogeny  
   Tyrosine‐protein kinase Fgr, encoded by the FGR (SRC2) gene (Uniprot ID: P09769), belongs to the Src family of non‐receptor tyrosine kinases. Fgr is evolutionarily conserved among vertebrates, and orthologs have been identified in mammals (including murine and human species) as well as in other higher eukaryotes. Within the kinome, Fgr is categorized together with members such as Src, Hck, Lyn, Fyn, Yes, and Blk. Its evolutionary relationships have been established based on sequence conservation and similar modular domain architectures that include the N‐terminal unique region, the SH3 and SH2 domains, and the catalytic kinase domain. Phylogenetic studies, including those by Manning et al. (2002, published in Science and Trends in Biochemical Sciences), indicate that Src family kinases originated from an early duplication event from a common ancestor of eukaryotes; Fgr represents a lineage specialized for regulation in hematopoietic cells (continolo2005theprotooncogenefgr pages 1-2, shen2018thesrcfamily pages 1-2).
2. Reaction Catalyzed  
   Fgr catalyzes the transfer of the terminal phosphate group from ATP to tyrosine residues on target proteins. The general chemical reaction is:  
    ATP + [protein]-L-tyrosine → ADP + [protein]-L-tyrosine-phosphate + H⁺  
   This reaction is characteristic of tyrosine kinases and is essential for transmitting intracellular signals that control a variety of cellular responses (du2022atpsiteinhibitorsinduce pages 3-5).
3. Cofactor Requirements  
   The catalytic activity of Fgr requires divalent cations to facilitate ATP binding and phosphate transfer. In common with other tyrosine kinases, Fgr is dependent on Mg²⁺ as a crucial cofactor. The presence of Mg²⁺ helps coordinate the phosphate groups of ATP within the kinase active site, thereby supporting phosphorylation reactions (du2022atpsiteinhibitorsinduce pages 3-5).
4. Substrate Specificity  
   Fgr phosphorylates tyrosine residues on a range of substrates implicated in immune and cytoskeletal signaling. Its substrate specificity is defined in part by its catalytic domain and the surrounding regulatory regions. Experimentally, Fgr has been shown to phosphorylate targets including the p85 regulatory subunit of phosphatidylinositol 3-kinase (PI3K), cortactin, focal adhesion kinase (FAK), and the Rac guanine nucleotide exchange factor Vav2 (continolo2005theprotooncogenefgr pages 1-2, continolo2005theprotooncogenefgr pages 14-15). In addition, studies have demonstrated that Fgr can phosphorylate SYK in vitro, thereby promoting downstream signaling (continolo2005theprotooncogenefgr pages 15-16). Although no single consensus motif has been definitively attributed to Fgr substrates, its intrinsic substrate specificity as a tyrosine kinase is consistent with findings reported for the human tyrosine kinome (Yaron-Barir2024 may be consulted for the comprehensive analysis of tyrosine kinase substrate preferences, Johnson2023 is relevant for serine/threonine kinases; both references contextualize the specificity characteristics within the broader kinase family) (shen2018thesrcfamily pages 2-3).
5. Structure  
   Fgr exhibits the classic domain organization characteristic of Src family kinases. Its structure is composed of an N‐terminal unique (SH4) domain that is post‐translationally modified by myristoylation and palmitoylation, ensuring proper membrane localization (continolo2005theprotooncogenefgr pages 12-14). Next, the protein contains an SH3 domain responsible for binding polyproline motifs, and an SH2 domain that recognizes phosphorylated tyrosine residues present in target proteins or within intramolecular sequences. Following these regulatory domains lies the central catalytic (kinase) domain, which contains an activation loop that can be phosphorylated on a tyrosine residue analogous to Tyr416 in Src. The C-terminal region harbors an inhibitory phosphorylation site (comparable to Tyr527 in Src) that, when phosphorylated by kinases such as C-terminal Src kinase (CSK), stabilizes an autoinhibited conformation (continolo2005theprotooncogenefgr pages 14-15, shen2018thesrcfamily pages 6-7). Recent crystallographic studies, particularly those using ATP-site inhibitors (A-419259 and TL02–59), have detailed that Fgr can adopt multiple conformations. In the presence of A-419259, Fgr is locked in a closed conformation with an outward rotation of the C-helix that disrupts the Glu310-Lys295 salt bridge; conversely, TL02–59 binding induces a type II conformation with the αC-helix rotated inward and the formation of the Glu310-Lys295 ion pair (du2022atpsiteinhibitorsinduce pages 14-18, du2022atpsiteinhibitorsinduce pages 18-23). These structures reveal both intramolecular dimerization interfaces and allosteric uncoupling of the regulatory SH3/SH2 domains from the catalytic core (du2022atpsiteinhibitorsinduce pages 23-26).
6. Regulation  
   Fgr activity is regulated by multiple post-translational modifications and intramolecular domain interactions. The N-terminal lipid modifications (myristoylation and palmitoylation) are essential for targeting Fgr to the plasma membrane, where it associates with receptor complexes, ensuring its proper spatial regulation (continolo2005theprotooncogenefgr pages 1-2, continolo2005theprotooncogenefgr pages 12-14). Phosphorylation of the activation loop—analogous to Tyr416 in Src—serves to activate the kinase by promoting a conformational change that aligns the catalytic residues, while phosphorylation of the C-terminal tail provides an inhibitory signal through binding to the SH2 domain (continolo2005theprotooncogenefgr pages 14-15, shen2018thesrcfamily pages 6-7). In specific cellular contexts, Fgr forms complexes with proteins such as focal adhesion kinase (FAK) and p190RhoGAP, which modulate downstream signaling involved in cytoskeletal rearrangement and cell migration (continolo2005theprotooncogenefgr pages 15-16, gresham2000negativeregulationof pages 1-2). Moreover, structural studies demonstrate that binding of ATP-competitive inhibitors can induce distinct conformational states that affect the regulatory interactions between the kinase and its SH domains (du2022atpsiteinhibitorsinduce pages 5-6, du2022atpsiteinhibitorsinduce pages 8-9). Fgr phosphorylation of its substrates is also influenced by PI3K-dependent mechanisms, and its activity can be modulated by extracellular signals, such as those mediated by integrins and immunoglobulin Fc receptors, which in turn alters the phosphorylation status of both Fgr and its interacting partners (continolo2005theprotooncogenefgr pages 1-2, vines2001inhibitionofβ2 pages 1-2).
7. Function  
   Fgr has diverse roles in immune cell signaling and cytoskeletal regulation. In hematopoietic cells—particularly in neutrophils, monocytes, macrophages, and mast cells—Fgr transduces signals from receptors that do not possess intrinsic kinase activity. In mast cells, Fgr is required for FcεRI-mediated signaling that results in Syk activation, subsequent phosphorylation of downstream adaptor proteins (including LAT, SLP76, and Gab2), and ultimately mast cell degranulation and cytokine release, contributing to IgE-mediated anaphylaxis (lee2011thesrcfamily pages 1-2, lee2011thesrcfamily pages 6-7). In macrophages and monocytes, Fgr plays a dual role where it can act as a positive regulator of migration by activating Rac and promoting actin cytoskeletal reorganization, while simultaneously serving as a negative regulator of integrin (ITGB2) signaling and phagocytosis through interactions with Syk and the recruitment of inhibitory phosphatases (gresham2000negativeregulationof pages 13-14, vines2001inhibitionofβ2 pages 7-9). Fgr also phosphorylates components of the focal adhesion complexes, including FAK and cortactin, linking adhesion dynamics to cell motility (continolo2005theprotooncogenefgr pages 15-16). In the context of hematological malignancies, particularly acute myeloid leukemia (AML), Fgr is frequently overexpressed and constitutively active; knockdown or pharmacological inhibition of Fgr in AML cell models leads to reduced cellular proliferation and tumor growth both in vitro and in vivo (du2022atpsiteinhibitorsinduce pages 1-3, weir2018selectiveinhibitionof pages 1-4). Thus, Fgr participates in signaling pathways downstream of immunoglobulin Fc receptors (MS4A2/FCER1B, FCGR2A/FCGR2B) and integrins (ITGB1 and ITGB2) to modulate cytoskeletal rearrangements, cell adhesion, migration, and inflammatory responses while its dysregulation has been implicated in oncogenesis and inflammation (continolo2005theprotooncogenefgr pages 1-2, lee2011thesrcfamily pages 7-8, shu2025constitutiveactivationof pages 1-2).
8. Other Comments  
   Recent studies have also identified small molecule inhibitors that target Fgr specifically. ATP-site inhibitors such as A-419259 and TL02–59 have been shown to induce unique conformations in Fgr, leading to potent suppression of its kinase activity in AML models (du2022atpsiteinhibitorsinduce pages 14-18, du2022atpsiteinhibitorsinduce pages 5-8). In addition, gain-of-function mutations in Fgr have been associated with autoinflammatory bone diseases in both mice and humans, with specific missense mutations (e.g., p.Arg118Trp, p.Asp502Gly) resulting in increased kinase activity and pathological bone inflammation (abe2019gainoffunctionmutationsin pages 3-3). Inhibitors of Fgr may hold clinical promise not only for AML but potentially for other immunologically mediated conditions. Fgr’s dual role as a positive regulator of certain pathways (e.g., mast cell degranulation and migration) and a negative regulator in integrin-dependent phagocytosis underscores the potential for context-dependent therapeutic strategies (continolo2005theprotooncogenefgr pages 15-16, vines2001inhibitionofβ2 pages 12-13).  
   Furthermore, Fgr has been shown to interact with a number of signaling molecules, including SYK, PIK3R1, PLD2, FAK, CBL, and VAV2, placing it at a nodal point in the regulation of immune receptor signaling, cytoskeletal dynamics and, ultimately, cellular motility and inflammatory responses (continolo2005theprotooncogenefgr pages 14-15, lee2011thesrcfamily pages 5-6). This broad involvement in multiple pathways makes Fgr a notable target for drug development, and ongoing studies continue to evaluate its substrate specificity within the tyrosine kinome (Yaron-Barir2024, Johnson2023). Its regulation by intracellular localization and tyrosine phosphorylation further adds layers of complexity that may be exploited by selective inhibitors (du2022atpsiteinhibitorsinduce pages 8-9, shen2018thesrcfamily pages 7-8).
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