1. Phylogeny  
   Tyrosine‐protein kinase Fgr is a member of the Src family kinases, which comprise a well‐conserved group of non‐receptor tyrosine kinases that arose early in eukaryotic evolution and are present in all metazoans. Fgr is closely related to other myeloid-specific Src family members such as Hck and Lyn and is predominantly expressed in hematopoietic cells including neutrophils, monocytes, macrophages, and mast cells (sen2011regulationofsrc pages 1-2, hatakeyama1994themurinecfgr pages 1-2). Phylogenetic analyses place Fgr within the core set of Src family kinases that descended from a common ancestor shared with other Src members, reflecting both conserved catalytic domains and divergent regulatory and N-terminal sequences that confer cell-specific roles (hatakeyama1994themurinecfgr pages 1-2, fumagalli2007thesrcfamily pages 1-2).
2. Reaction Catalyzed  
   Fgr catalyzes the phosphorylation of tyrosine residues on target proteins by transferring the γ-phosphate from ATP to the hydroxyl group of a tyrosine residue in substrate proteins. In precise biochemical terms, the catalytic reaction is described as: ATP + [protein]-L-tyrosine → ADP + [protein]-L-tyrosine-phosphate + H⁺ (kemble2009abiochemicalstudy pages 146-150).
3. Cofactor Requirements  
   The kinase activity of Fgr, like that of other Src family kinases, is dependent on the presence of Mg²⁺ ions. Magnesium acts as an essential cofactor by coordinating the ATP substrate and stabilizing the transition state during the phosphoryl transfer reaction (kemble2009abiochemicalstudy pages 146-150).
4. Substrate Specificity  
   Fgr preferentially phosphorylates tyrosine residues within substrates that are functionally recognized through phosphotyrosine motifs. Studies of Src family kinases have demonstrated that substrate recognition is mediated by the kinase’s SH2 domain, which interacts with pre-phosphorylated motifs, thereby facilitating secondary phosphorylation events. For example, Fgr has been shown to phosphorylate HS1 only after priming phosphorylation by Syk kinase, with specific targeting of the Tyr-222 residue (brunati1999molecularfeaturesunderlying pages 1-2, brunati1999molecularfeaturesunderlying pages 4-6). In addition, Fgr phosphorylates several signaling intermediates that contain consensus sequences similar to those recognized by other Src kinases, although an explicit consensus motif for Fgr alone has not been universally defined (brunati1999molecularfeaturesunderlying pages 6-7).
5. Structure  
   Fgr exhibits the typical domain architecture of Src family kinases, which includes an N-terminal unique region that undergoes myristoylation, an SH3 domain, an SH2 domain, a conserved catalytic kinase domain, and a short C-terminal regulatory region. The N-terminal region, which is less conserved compared to the catalytic core, targets Fgr to membranes through myristoylation and helps determine subcellular localization (hatakeyama1994themurinecfgr pages 3-3, sen2011regulationofsrc pages 1-2). The SH3 domain mediates interactions with proline-rich motifs in substrates and regulatory proteins, while the SH2 domain binds to phosphorylated tyrosine residues in specific sequence contexts to regulate both substrate targeting and intramolecular interactions. The kinase domain contains the catalytic cleft, with conserved residues constituting the activation loop and C-helix that modulate kinase activity through conformational changes and autophosphorylation (kemble2009abiochemicalstudy pages 155-159, hatakeyama1994themurinecfgr pages 3-4). Unique structural features of Fgr include its distinct nonhomologous N-terminal segment and a C-terminal tail that is critical for negative regulation by phosphorylation. These features collectively contribute to both the catalytic efficiency and the regulatory control of Fgr (hatakeyama1994themurinecfgr pages 4-5, kemble2009abiochemicalstudy pages 29-33).
6. Regulation  
   Fgr is subjected to a variety of regulatory mechanisms that modulate its kinase activity. Autophosphorylation events within the kinase domain are a central part of activation, while phosphorylation of a conserved C-terminal tyrosine residue by C-terminal Src kinase (Csk) mediates negative regulation by promoting an inactive conformation through intramolecular SH2 domain binding (ruzzene1994regulationofcfgr pages 1-2, kemble2009abiochemicalstudy pages 155-159). In addition, gain-of-function mutations, such as the p.Asp502Gly substitution identified in both murine models and human patients afflicted with autoinflammatory bone disease, alter the regulatory phosphorylation dynamics and disrupt the balance of kinase signaling (abe2019gainoffunctionmutationsin pages 1-1, abe2019gainoffunctionmutationsin pages 2-3). Fgr is also modulated through interactions with adaptor proteins and through its association with cell-surface receptors, such as Fc receptors, where receptor crosslinking triggers enhanced autophosphorylation and catalytic activity (toyoshima1993associationofimmunoglobulin pages 1-2, toyoshima1993associationofimmunoglobulin pages 4-5). Moreover, studies in monocytes have shown that Fgr can interact with Syk kinase via its SH2 domain, leading to inhibition of Syk-dependent signaling pathways in β2 integrin receptor signaling (vines2001inhibitionofβ2 pages 13-13, vines2001inhibitionofβ2 pages 6-7). Collectively, these modifications—mediated by upstream kinases such as Csk, by autophosphorylation, and through protein–protein interactions—allow tight spatial and temporal control over Fgr activity (ruzzene1994regulationofcfgr pages 4-6, abe2019gainoffunctionmutationsin pages 5-6).
7. Function  
   Fgr plays a multifaceted role in immune cell signaling by transmitting signals from cell surface receptors that lack intrinsic kinase activity. It is expressed primarily in hematopoietic cells, including neutrophils, monocytes, macrophages, and mast cells, where it regulates critical responses such as cell adhesion, migration, phagocytosis, and cytoskeletal remodeling. In neutrophils, Fgr is essential for the proper activation of the respiratory burst and for actin cytoskeleton reorganization in response to chemotactic peptides such as fMLP, as it contributes to the phosphorylation of key intermediates like Vav1 and downstream activation of Rac GTPases, which in turn regulate NADPH oxidase activity (fumagalli2007thesrcfamily pages 11-12, fumagalli2007thesrcfamily pages 8-9). In monocytes, Fgr has a dual role; while it supports ITGB1 and ITGB2-mediated signaling required for cell spreading and adhesion, it can function as a negative regulator of β2 integrin signaling and Syk kinase activity under specific conditions, thereby modulating phagocytosis and integrin-mediated cytoskeletal rearrangements (jing2021genedeficiencyor pages 11-12, vines2001inhibitionofβ2 pages 2-3). In the context of mast cells, Fgr contributes to degranulation and the release of inflammatory cytokines by phosphorylating PLD2, which leads to the generation of secondary lipid messengers such as lysophosphatidic acid and diacylglycerol (jing2021genedeficiencyor pages 12-12). Additionally, Fgr phosphorylates several other substrates—including CBL, VAV2, PTK2/FAK1, and HCLS1—thereby linking it to signaling cascades that influence cell survival, proliferation, and migration (fumagalli2007thesrcfamily pages 5-6, weir2018selectiveinhibitionof pages 11-14). Recent studies in acute myeloid leukemia (AML) have revealed that Fgr is overexpressed in certain patient subsets and that selective inhibition of Fgr with compounds such as TL02-59 leads to potent suppression of AML cell growth in vitro and in vivo (weir2018selectiveinhibitionof pages 11-14, weir2018selectiveinhibitionof pages 4-8). Hence, Fgr is implicated in both normal immune regulation and in pathological conditions, such as autoinflammatory bone disease, AML, and possibly other immune-related disorders (abe2019gainoffunctionmutationsin pages 1-1, abe2019gainoffunctionmutationsin pages 2-3).
8. Other Comments  
   Experimental inhibitors targeting Src family kinases, including Fgr, have been developed; notably, the N-phenylbenzamide compound TL02-59 exhibits picomolar potency against Fgr and has been shown to effectively suppress AML cell proliferation, highlighting the therapeutic potential of selective Fgr inhibition (weir2018selectiveinhibitionof pages 8-11, weir2018selectiveinhibitionof pages 11-14). Gain-of-function mutations in Fgr, such as the p.Asp502Gly substitution, have been linked to autoinflammatory bone disease in both murine models and human patients, establishing Fgr as a candidate susceptibility gene in these disorders (abe2019gainoffunctionmutationsin pages 1-1, abe2019gainoffunctionmutationsin pages 2-3). In addition, Fgr has been implicated in pathways downstream of Fc receptors, where it modulates immune responses such as IgE-mediated anaphylaxis and cytokine release in mast cells, thus further linking its activity to inflammatory and immune-mediated conditions (toyoshima1993associationofimmunoglobulin pages 1-2, jing2021genedeficiencyor pages 12-12). The kinase’s regulation of integrin signaling in both neutrophils and monocytes underscores its importance in modulating cell adhesion, spreading, and transmigration, processes that are critical during immune surveillance and inflammation (fumagalli2007thesrcfamily pages 9-10, vines2001inhibitionofβ2 pages 5-6). These multiple functional roles, coupled with its conserved domain structure and dynamic regulation by phosphorylation and protein–protein interactions, designate Fgr as a key node in immune cell signaling with significant implications for therapeutic intervention in cancer and autoinflammatory disorders (fumagalli2007thesrcfamily pages 11-12, abe2019gainoffunctionmutationsin pages 5-6).

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