1. Phylogeny  
   FGFR1 is a member of the fibroblast growth factor receptor family, which comprises four main receptor tyrosine kinases (FGFR1–FGFR4) and an additional receptor‐like protein (FGFRL1) that lacks a catalytic domain, and these receptors are evolutionarily conserved across vertebrate species (ornitz2015thefibroblastgrowth pages 1-2, beenken2009thefgffamily pages 2-4, patani2018assessmentoffibroblast pages 13-17). FGFR1 orthologs have been identified in all mammalian species, and the conservation extends to non-mammalian vertebrates, underscoring its fundamental role in processes such as embryogenesis and tissue homeostasis (ornitz2015thefibroblastgrowth pages 1-2, patani2018assessmentoffibroblast pages 13-17). Comparative sequence analyses reveal that the extracellular ligand-binding domains as well as the intracellular tyrosine kinase domain exhibit a high degree of conservation, indicative of stringent evolutionary constraints on the functional regions necessary for ligand recognition and catalytic activity (beenken2009thefgffamily pages 2-4, patani2018assessmentoffibroblast pages 13-17). Moreover, the mechanisms underlying alternative splicing, particularly within the third immunoglobulin-like (Ig) domain that generates the FGFR1b and FGFR1c isoforms, are maintained among vertebrates, thereby providing differential ligand-binding capabilities and tissue-specific signaling outputs (tabatabaei2022introductionofhuman pages 25-29, patani2018assessmentoffibroblast pages 27-30). Phylogenetic studies situate FGFR1 within the receptor tyrosine kinase superfamily, sharing a common evolutionary origin with other kinases that emerged in the Last Eukaryotic Common Ancestor (LECA), and it is part of a highly conserved core set of kinases that includes PI3K, AKT, and MAPKs (ornitz2015thefibroblastgrowth pages 1-2, beenken2009thefgffamily pages 2-4). This evolutionary conservation, along with the preservation of key structural motifs, such as the conserved DFG motif and catalytic loop found in the kinase domain, reflects the critical biological functions that FGFR1 fulfills from early development through adulthood (farrell2018structureactivationand pages 1-2, dai2019fibroblastgrowthfactor pages 4-5). In summary, FGFR1’s phylogenetic footprint is marked by its wide conservation across species, its membership in the FGFR subfamily of RTKs, and its retention of conserved domain architectures that underlie essential signaling roles (ornitz2015thefibroblastgrowth pages 1-2, beenken2009thefgffamily pages 2-4, patani2018assessmentoffibroblast pages 13-17).
2. Reaction Catalyzed  
   FGFR1 functions as a receptor tyrosine kinase that catalyzes the transfer of a phosphate group from ATP to specific tyrosine residues on substrate proteins, including itself via autophosphorylation (dai2019fibroblastgrowthfactor pages 8-10, roskoski2020theroleof pages 1-7). The chemical reaction it mediates can be summarized as follows: ATP + [protein]-tyrosine → ADP + [protein]-phosphotyrosine + H⁺, where the substrate may be the receptor itself or downstream adaptor proteins that convey the signal (roskoski2020theroleof pages 1-7, dai2019fibroblastgrowthfactor pages 8-10). This phosphorylation event acts as a molecular switch that leads to the activation of various intracellular signaling cascades involved in cellular proliferation, differentiation, migration, and survival (agrawal2020designofhfgf1 pages 29-33, dai2019fibroblastgrowthfactor pages 10-12).
3. Cofactor Requirements  
   The catalytic activity of FGFR1 is dependent on the presence of ATP and, critically, on divalent metal ions—most notably magnesium (Mg²⁺)—which help to coordinate ATP binding within the kinase active site (dai2019fibroblastgrowthfactor pages 1-4, roskoski2020theroleof pages 7-11). The Mg²⁺ ion interacts with the phosphate groups of ATP, facilitating the correct orientation of the nucleotide for efficient phosphoryl transfer (dai2019fibroblastgrowthfactor pages 1-4, roskoski2020theroleof pages 7-11). Accordingly, the presence of sufficient Mg²⁺ is essential for FGFR1 to catalyze its phosphorylation reaction effectively.
4. Substrate Specificity  
   FGFR1 demonstrates substrate specificity characteristic of receptor tyrosine kinases by preferentially phosphorylating tyrosine residues within defined regions of its own intracellular domain and on associated downstream signaling proteins (agrawal2020designofhfgf1 pages 29-33, dai2019fibroblastgrowthfactor pages 10-12). Key autophosphorylation sites on FGFR1 include tyrosine residues Y653 and Y654, which are located in the activation loop of the kinase domain and are essential for enhancing catalytic activity upon phosphorylation (agrawal2020designofhfgf1 pages 29-33, roskoski2020theroleof pages 7-11). In addition, FGFR1 phosphorylates substrates such as PLCγ1, FRS2, GAB1, and SHB, whose docking and subsequent binding rely on the formation of phosphotyrosine-containing motifs that are recognized by specific SH2 domains (roskoski2020theroleof pages 1-7, agrawal2020designofhfgf1 pages 29-33). Although a precise linear consensus phosphorylation motif for FGFR1 substrates has not been universally defined, the context of amino acid residues surrounding the tyrosine and the spatial conformation of the substrate are critical determinants of substrate specificity (dai2019fibroblastgrowthfactor pages 10-12, roskoski2020theroleof pages 1-7).
5. Structure  
   FGFR1 is a multi-domain protein that exhibits a modular structure, with distinct regions contributing to its receptor and catalytic functions. The extracellular region consists of three immunoglobulin-like (Ig) domains, designated D1, D2, and D3; the D1 domain contains an acidic “acid box” that contributes to autoinhibition by modulating ligand binding, while the D2 and D3 domains form the ligand-binding interface and are responsible for engaging fibroblast growth factors (FGFs) and heparan sulfate proteoglycans (beenken2009thefgffamily pages 2-4, tabatabaei2022introductionofhuman pages 25-29). Alternative splicing within the third Ig domain generates isoforms such as FGFR1b and FGFR1c that differ in ligand-binding specificity and tissue distribution (tabatabaei2022introductionofhuman pages 25-29, patani2018assessmentoffibroblast pages 27-30). Following the extracellular region is a single transmembrane domain that anchors the receptor in the plasma membrane and facilitates dimerization upon ligand engagement (beenken2009thefgffamily pages 4-6, ornitz2015thefibroblastgrowth pages 2-4). The intracellular portion of FGFR1 is composed of a split tyrosine kinase domain, which displays a bilobed structure typical of protein kinases; the N-terminal lobe is characterized by a glycine-rich loop (P-loop) that is crucial for ATP binding, while the C-terminal lobe contains the catalytic loop, the activation loop (A-loop) with conserved tyrosine residues such as Y653 and Y654, and a DFG motif that toggles between active (DFG-in) and inactive (DFG-out) conformations (dai2019fibroblastgrowthfactor pages 4-5, farrell2018structureactivationand pages 4-6). In addition, the kinase domain contains a hydrophobic regulatory spine and an αC-helix that plays a key role in the regulation of catalytic activity; mechanisms such as the “molecular brake” further stabilize the receptor’s inactive conformation until ligand-induced dimerization permits its release and subsequent activation (farrell2018structureactivationand pages 15-16, dai2019fibroblastgrowthfactor pages 8-10, roskoski2020theroleof pages 56-73). Collectively, this structural organization enables FGFR1 to couple extracellular ligand recognition with intracellular kinase activation in a highly regulated manner (beenken2009thefgffamily pages 2-4, ornitz2015thefibroblastgrowth pages 27-29).
6. Regulation  
   The regulation of FGFR1 is achieved through a combination of extracellular, intramolecular, and post-translational mechanisms. In the absence of ligand, the receptor is maintained in an autoinhibited state by intra-domain interactions, notably involving the D1 domain and the “acid box,” which hinder ligand binding and prevent spontaneous receptor activation (agrawal2020designofhfgf1 pages 29-33, beenken2009thefgffamily pages 2-4). Ligand binding, in conjunction with the cofactor heparan sulfate, induces receptor dimerization and brings the intracellular kinase domains into close proximity, thereby initiating trans-autophosphorylation on critical tyrosine residues including Y653 and Y654; this step significantly increases kinase activity and creates docking sites for downstream signaling molecules (agrawal2020designofhfgf1 pages 25-29, dey2010investigatingthecontribution pages 17-20). Further regulation is provided by negative feedback mechanisms: adaptor proteins such as GRB2 and regulatory molecules like Sprouty proteins are recruited upon receptor activation to attenuate signaling intensity, while ubiquitin ligases (for example, Cbl) target the receptor for endocytosis and degradation to limit the duration of the signal (dey2010investigatingthecontribution pages 20-24, patani2018assessmentoffibroblast pages 211-213). Conformational changes within the kinase domain—such as the repositioning of the αC-helix and alignment of the hydrophobic spines—are integral to shifting FGFR1 from an inactive to an active conformation (farrell2018structureactivationand pages 15-16, dai2019fibroblastgrowthfactor pages 8-10). This tightly choreographed regulation is essential for ensuring that FGFR1-mediated signaling occurs only in the appropriate cellular context and with the proper intensity (roskoski2020theroleof pages 7-11, dey2010investigatingthecontribution pages 35-39).
7. Function  
   FGFR1 functions primarily as a cell-surface receptor for fibroblast growth factors and plays a pivotal role in numerous developmental and physiological processes. During embryogenesis, FGFR1 signaling is required for normal mesoderm patterning, axial organization, and skeletogenesis, and it is crucial for the proper development of the gonadotropin-releasing hormone (GnRH) neuronal system (agrawal2020designofhfgf1 pages 52-55, ornitz2015thefibroblastgrowth pages 1-2). Upon binding FGFs in the presence of heparan sulfate, FGFR1 dimerizes and activates its intrinsic tyrosine kinase domain, leading to autophosphorylation and subsequent recruitment of adaptor proteins such as FRS2, PLCγ1, GAB1, and SHB; these adaptor proteins mediate downstream activation of critical signaling cascades including the RAS/MAPK, PI3K/AKT, and PLCγ pathways (dai2019fibroblastgrowthfactor pages 10-12, agrawal2020designofhfgf1 pages 29-33, roskoski2020theroleof pages 1-7). These signaling pathways regulate cell proliferation, differentiation, migration, and survival and are essential for processes ranging from tissue repair and angiogenesis to metabolic homeostasis (ornitz2015thefibroblastgrowth pages 1-2, patani2018assessmentoffibroblast pages 27-30). FGFR1 is expressed in a wide variety of tissues, with its alternative splicing generating receptor isoforms that enable context-dependent responses to different FGF ligands, thereby fine-tuning cellular responses during both development and adult physiology (tabatabaei2022introductionofhuman pages 25-29, patani2018assessmentoffibroblast pages 27-30). Aberrant FGFR1 activity—whether due to overexpression, gene amplification, or gain-of-function mutations—has been implicated in several cancers including breast, lung, and prostate carcinomas, underscoring its clinical significance as both a biomarker and a therapeutic target (agrawal2020designofhfgf1 pages 37-41, zhang2019targetingtheoncogenic pages 1-3, tanner2019drugresistancemechanisms pages 26-30).
8. Other Comments  
   FGFR1 is the subject of extensive research not only because of its central biological functions but also due to its relevance in disease pathology and therapeutic intervention. A number of small-molecule inhibitors, such as erdafitinib and AZD4547, have been developed to target the ATP-binding pocket of FGFR1 and are currently undergoing clinical evaluation for the treatment of FGFR-driven cancers (roskoski2020theroleof pages 87-91, turner2018towardsselectiveinhibitors pages 272-274). Resistance to such inhibitors can emerge through mutations within the kinase domain, including alterations at the gatekeeper residue, leading to reduced drug binding and therapeutic efficacy; these observations have spurred efforts to design next-generation inhibitors that can overcome resistance mechanisms (tanner2019drugresistancemechanisms pages 26-30, roskoski2020theroleof pages 43-47). In addition, FGFR1 dysregulation has been associated with several developmental disorders, such as Pfeiffer syndrome and Kallmann syndrome, which highlights the receptor’s critical role in both normal development and disease (agrawal2020designofhfgf1 pages 37-41, dey2010investigatingthecontribution pages 35-39). The ability of FGFR1 to transduce signals from the extracellular environment to the nucleus—thereby influencing transcription factors like CREB1 and RPS6KA1—further broadens its impact on cellular function and underscores the complexity of its signaling network (roskoski2020theroleof pages 7-11, patani2018assessmentoffibroblast pages 216-218). Overall, the multifaceted regulation and diverse functional roles of FGFR1, coupled with its involvement in a broad spectrum of diseases, continue to make it a highly attractive target in biomedical research and drug discovery (agrawal2020designofhfgf1 pages 25-29, ornitz2015thefibroblastgrowth pages 52-52).
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