1. Phylogeny  
   Vascular endothelial growth factor receptor 1 (VEGFR1), encoded by the FLT1 gene and commonly referred to as Flt‑1, is a receptor tyrosine kinase (RTK) that belongs to the VEGFR subfamily within the larger kinome. It is evolutionarily conserved among vertebrates, with orthologous forms identified in a wide range of species from lower vertebrates to mammals. VEGFR1 shares substantial sequence and structural similarity with its family members VEGFR2 (KDR/Flk‑1) and VEGFR3 (Flt‑4); however, it diverges functionally by exhibiting distinct ligand‐binding characteristics and a low intrinsic kinase activity. Comparative analyses place VEGFR1 within the type III RTK group, and its conservation of multiple immunoglobulin-like domains in the extracellular region underscores its essential role in vascular development and angiogenic regulation (cebesuarez2006theroleof pages 1-2, guo2010vascularendothelialgrowth pages 1-2).
2. Reaction Catalyzed  
   The catalytic reaction mediated by VEGFR1 involves the transfer of the γ‐phosphate from ATP to specific tyrosine residues on substrate proteins. In its role as a tyrosine kinase, VEGFR1 carries out the following chemical conversion: ATP + [protein]-tyrosine → ADP + [protein]-tyrosine-phosphate + H⁺. This reaction is initiated by ligand binding, which leads to receptor dimerization and autophosphorylation of key tyrosine residues within its cytoplasmic domain, thereby triggering downstream signaling cascades (matsumoto2001vegfreceptorsignal pages 2-3, asthana2019structuralandfunctional pages 29-33).
3. Cofactor Requirements  
   The kinase activity of VEGFR1 is dependent on divalent metal ions; in particular, Mg²⁺ ions are essential for ATP binding and the subsequent phosphotransfer reaction. This requirement for Mg²⁺ is a hallmark of kinases, ensuring proper positioning and stabilization of the ATP molecule within the active site during catalysis (dai2007vegfanessential pages 4-6, matsumoto2001vegfreceptorsignal pages 2-3).
4. Substrate Specificity  
   VEGFR1 functions primarily through autophosphorylation, whereby its own cytoplasmic tyrosine residues serve as substrates for its kinase activity following receptor dimerization. Specific phosphorylation events occur on residues such as Tyr1169 and Tyr1213, which then create docking sites for intracellular signaling effectors including the p85 subunit of phosphatidylinositol 3‑kinase, phospholipase Cγ (PLCγ), the SH2-domain–containing phosphatase SHP2, and the adaptor protein Grb2. Although a defined consensus phosphorylation motif for VEGFR1 is less well established compared with some other RTKs, these targeted autophosphorylation events are critical for mediating its functions in modulating angiogenic signal transduction (asthana2019structuralandfunctional pages 29-33, failla2018positiveandnegative pages 10-12, kreuger2006vegfreceptorsignalling?a pages 1-2).
5. Structure  
   VEGFR1 is organized into three major regions: an extracellular domain, a single transmembrane segment, and an intracellular tyrosine kinase domain. The extracellular portion is composed of seven immunoglobulin-like (Ig-like) domains, of which domains 2 and 3 play a crucial role in high-affinity binding of VEGF ligands such as VEGF-A, VEGF-B, and placental growth factor (PlGF); these domains form the primary ligand-binding interface (astahana2019structuralandfunctional pages 29-33, kinghorn2023vesicletraffickingand pages 27-31). The transmembrane domain anchors the receptor in the plasma membrane, while the intracellular region contains a split tyrosine kinase domain interrupted by a kinase-insert segment. This kinase domain harbors catalytic motifs necessary for ATP binding and phosphotransfer, yet its activity is lower relative to VEGFR2. Structural analyses indicate that this attenuated catalytic function may be attributed to a unique substitution within the activation loop and the presence of an inhibitory juxtamembrane sequence that dampens kinase activity (astahana2019structuralandfunctional pages 145-147, shibuya2010tyrosinekinasereceptor pages 2-3, rittler2008modelingofinteractions pages 14-17). In addition, alternative splicing of the FLT1 transcript gives rise to soluble isoforms (sVEGFR1) that lack the transmembrane and intracellular regions; these isoforms are capable of binding VEGF ligands with an affinity comparable to the full-length receptor, thereby functioning as potent decoys to regulate ligand availability (failla2018positiveandnegative pages 3-5, astahana2019structuralandfunctional pages 29-33). High-resolution structural studies, including crystallographic analyses and advanced in silico models, have provided insights into the domain organization and the ligand-induced conformational changes that underlie receptor dimerization, autophosphorylation, and subsequent signal initiation (shaik2020structuralbasisfor pages 4-6, shaik2020structuralbasisfor pages 6-7).
6. Regulation  
   VEGFR1 is regulated by multiple mechanisms that encompass post-translational modifications, receptor dimerization, and alternative RNA splicing. Ligand binding induces receptor dimerization, which is a prerequisite for autophosphorylation of specific cytoplasmic tyrosine residues (such as Tyr1169 and Tyr1213); these phosphorylation events serve as key regulatory switches that facilitate the recruitment of downstream signaling molecules (astahana2019structuralandfunctional pages 29-33, shibuya2001structureandfunction pages 10-11). A notable feature of VEGFR1 regulation is the presence of a repressor sequence within its juxtamembrane region that curtails its tyrosine kinase activity, thereby ensuring that the receptor’s catalytic function remains subdued relative to that of VEGFR2 (astahana2019structuralandfunctional pages 145-147, shibuya2010tyrosinekinasereceptor pages 2-3). Furthermore, alternative splicing events result in the generation of soluble VEGFR1 isoforms (sVEGFR1), which modulate angiogenic stimuli by sequestering VEGF ligands away from cell-surface receptors, ultimately fine-tuning the balance of angiogenic signaling (failla2018positiveandnegative pages 3-5, rahimi2009identificationofligandinduced pages 1-2). In certain cellular contexts, ligand-induced proteolytic cleavage and receptor ectodomain shedding have been observed, adding an additional layer of regulation by modulating receptor availability at the cell surface (rahimi2009identificationofligandinduced pages 1-2). Collectively, these regulatory mechanisms maintain tight control over VEGFR1 signaling under both physiological and pathological conditions (kreuger2006vegfreceptorsignalling?a pages 3-4, matsumoto2001vegfreceptorsignal pages 3-4).
7. Function  
   VEGFR1 plays multiple roles in vascular biology, with functions that are both context dependent and cell-type specific. During embryogenesis, VEGFR1 has been shown to act predominantly as a negative regulator of angiogenesis. By sequestering VEGF-A with very high affinity, it limits excessive endothelial cell proliferation and prevents the formation of disorganized vascular networks. This role is underscored by knockout studies in which deletion of VEGFR1 leads to embryonic lethality due to unchecked vascular overgrowth (ferrara2001roleofvascular pages 2-3, shibuya2001structureandfunction pages 10-11, rahimi2006vascularendothelialgrowth pages 13-15). In contrast, in postnatal and adult tissues, VEGFR1 contributes positively to angiogenesis by promoting endothelial cell proliferation, survival, and migration in response to placental growth factor (PlGF) and certain VEGF-A isoforms. These functions are critical for vascular remodeling, tissue repair, and the regression of postnatal retinal hyaloid vessels (astahana2019structuralandfunctional pages 145-147, guo2010vascularendothelialgrowth pages 17-19). Moreover, VEGFR1 is expressed on cell types such as monocytes, macrophages, and hematopoietic stem cells, where it influences chemotaxis and inflammatory responses. In the setting of cancer, VEGFR1-mediated signaling has been implicated in tumor angiogenesis and cancer cell invasion, underscoring its role in modulating the tumor microenvironment (failla2018positiveandnegative pages 1-3, kreuger2006vegfreceptorsignalling?a pages 1-2, perezgutierrez2023biologyandtherapeutic pages 5-6). Thus, VEGFR1 functions as a critical mediator by balancing VEGF ligand bioavailability through its decoy activity and transmitting weaker, yet functionally significant, intracellular signals that modulate vascular development and pathological angiogenesis (astahana2019structuralandfunctional pages 139-142, ferrara2001roleofvascular pages 2-3).
8. Other Comments  
   Beyond its membrane-bound form, the FLT1 gene gives rise to soluble isoforms of VEGFR1 that serve as endogenous inhibitors of angiogenesis by trapping VEGF ligands. These soluble receptors are of considerable interest in clinical contexts, as elevated levels of sVEGFR1 have been implicated in the pathogenesis of preeclampsia and in the modulation of tumor angiogenesis. Therapeutically, numerous strategies targeting the VEGF/VEGFR axis have been developed; these include engineered fusion proteins, monoclonal antibodies, and small-molecule tyrosine kinase inhibitors designed to inhibit VEGFR1 activity or disrupt its interaction with VEGF ligands (lee2025vascularendothelialgrowth pages 28-29, guo2010vascularendothelialgrowth pages 17-19). Additionally, VEGFR1’s role in mediating macrophage migration and chemotaxis links it to inflammatory responses and cancer metastasis, further highlighting its relevance as a therapeutic target. The relatively low intrinsic kinase activity of VEGFR1, combined with its high ligand-binding affinity, underscores its dual functionality as both a modulator of VEGF availability and as a signaling receptor with cell-type–specific proliferative and migratory effects (failla2018positiveandnegative pages 1-3, rahimi2006vascularendothelialgrowth pages 20-21).
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