1. Phylogeny  
   Platelet‐derived growth factor receptor beta (PDGFRB, UniProt P09619) is a receptor tyrosine kinase that belongs to the type III receptor tyrosine kinase family. It shares an evolutionary background with other receptors in this family such as KIT, FLT3 and the macrophage‐colony stimulating factor receptor. Sequence comparisons and domain‐structure analyses show that PDGFRB is highly conserved across vertebrate species, indicating that its modular architecture—characterized by multiple extracellular immunoglobulin‐like (Ig) domains, a single spanning transmembrane helix and an intracellular tyrosine kinase domain—was established early during metazoan evolution (betsholtz2001developmentalrolesof pages 1-2, chen2013plateletderivedgrowthfactors pages 1-2). Phylogenetic studies have grouped PDGFRB with receptors that share similar ligand recognition mechanisms, and its evolutionary relationship with vascular endothelial growth factor receptors reflects the conservation of cysteine‐rich ligand‐binding motifs among these kinases (betsholtz2001developmentalrolesof pages 1-2, chen2013plateletderivedgrowthfactors pages 1-2).
2. Reaction Catalyzed  
   PDGFRB is an enzyme that catalyzes the transfer of a phosphate group from ATP to specific tyrosine residues on substrate proteins. The overall reaction can be summarized as follows: ATP + [protein]–L-tyrosine → ADP + [protein]–phospho-L-tyrosine + H⁺. This phosphorylation event modifies downstream signaling proteins by creating phosphotyrosine docking sites for SH2 domain-containing effectors, thereby propagating intracellular signals (kazlauskas1992gtpaseactivatingproteinand pages 1-2).
3. Cofactor Requirements  
   The intrinsic kinase activity of PDGFRB is dependent on divalent cations, with magnesium (Mg²⁺) acting as an essential cofactor. Mg²⁺ coordinates with ATP in the active site to facilitate the transfer of the γ-phosphate to the substrate tyrosine residues. Although manganese (Mn²⁺) may also be capable of supporting kinase activity under some conditions, Mg²⁺ is predominantly required for optimal catalytic function (kazlauskas1992gtpaseactivatingproteinand pages 1-2).
4. Substrate Specificity  
   PDGFRB phosphorylates tyrosine residues on target proteins; however, despite extensive research, a strict consensus sequence for its substrates has not been unequivocally defined. Instead, the receptor’s substrate specificity is dictated by the creation of phosphotyrosine docking sites that recruit specific SH2 domain-containing signaling molecules, including phospholipase C-γ (PLC-γ), the p85 subunit of phosphatidylinositol 3-kinase (PI3K) and GTPase activating proteins (GAPs). Binding studies have indicated that the local amino acid context surrounding the phosphorylated tyrosine can determine the affinity for different downstream effectors, yet common features often include flanking acidic or hydrophobic residues that promote recognition by the SH2 domains of these adaptor proteins (valius1993tyrosines1021and pages 1-2, kazlauskas1992gtpaseactivatingproteinand pages 1-2).
5. Structure  
   PDGFRB is organized into several distinct regions. The extracellular domain comprises five immunoglobulin-like (Ig) domains (designated D1 through D5) that mediate ligand binding; studies have highlighted that domains D1–D3 form the primary interaction surface for PDGF ligands, while domains D4 and D5 contribute to receptor dimerization through rigid hinge-like arrangements and salt bridge formation (chen2013plateletderivedgrowthfactors pages 4-5, chen2013plateletderivedgrowthfactors pages 7-8). Immediately following the extracellular region, a single-pass transmembrane helix anchors the receptor to the plasma membrane. The intracellular portion begins with a juxtamembrane segment that plays a critical role in autoinhibition; upon ligand binding and receptor dimerization, conformational changes relieve this autoinhibition. The intracellular domain is characterized by a split tyrosine kinase domain separated by a kinase insert region that contains multiple autophosphorylation sites including tyrosines Y740, Y751, Y771, and Y857, as well as additional C-terminal phosphorylation sites such as Y1009 and Y1021. These phosphorylated residues act as docking sites for downstream signaling proteins (kazlauskas1992gtpaseactivatingproteinand pages 1-2, valius1993tyrosines1021and pages 9-10). Structural studies, including those based on crystallography and high-confidence AlphaFold models, indicate that the receptor adopts a globular kinase domain with the characteristic C-helix, activation loop and hydrophobic spine required for catalytic activity. Unique features, such as heavy N-linked glycosylation sites on the extracellular Ig domains, contribute to receptor stability and ligand-binding specificity (chen2013plateletderivedgrowthfactors pages 4-5, chen2013plateletderivedgrowthfactors pages 8-10).
6. Regulation  
   Regulatory mechanisms of PDGFRB converge on its controlled activation by ligand-induced dimerization and subsequent autophosphorylation. Ligand binding (typically by PDGF-BB, PDGF-D for homodimeric receptors, or PDGF-AB in heterodimeric complexes with PDGFRα) induces a conformational change that promotes receptor dimerization; this juxtaposition enables reciprocal trans-autophosphorylation of tyrosine residues in the intracellular domain (betsholtz2001developmentalrolesof pages 2-4, chen2013plateletderivedgrowthfactors pages 10-11). Specific phosphorylation events—such as those at Y1021 and Y1009—are required for the stable recruitment of PLC-γ and a 64-kDa protein factor, respectively, which serve as key steps in downstream signal propagation (valius1993tyrosines1021and pages 1-2, valius1993tyrosines1021and pages 11-11). In addition, the juxtamembrane region undergoes regulatory phosphorylation that modulates the receptor’s kinase activity. PDGFRB activity is further fine-tuned by mechanisms of receptor internalization, ubiquitination, and subsequent lysosomal degradation, all of which serve to downregulate signaling after ligand stimulation (maudsley2000plateletderivedgrowthfactor pages 10-11, maudsley2000plateletderivedgrowthfactor pages 11-11). Moreover, associations with adaptor proteins such as the Na⁺/H⁺ exchanger regulatory factor (NHERF) can potentiate receptor activity by promoting the oligomerization of receptor complexes and linking the receptor to the actin cytoskeleton (maudsley2000plateletderivedgrowthfactor pages 10-11).
7. Function  
   PDGFRB functions as a critical mediator of multiple biological processes during both embryonic development and adult tissue homeostasis. It is predominantly expressed on cells of mesenchymal origin—including vascular smooth muscle cells, pericytes and fibroblasts—and is vital for cell proliferation, survival, migration, differentiation, and chemotaxis. During embryogenesis, PDGFRB signaling is essential for blood vessel development by promoting the proliferation, migration, and recruitment of pericytes and smooth muscle cells to nascent endothelial tubes; such interactions underlie the formation and stabilization of a branched capillary network that is crucial for proper organ function (betsholtz2001developmentalrolesof pages 13-14, chen2013plateletderivedgrowthfactors pages 1-2). In the kidney glomerulus, PDGFRB regulates the recruitment and maintenance of mesangial cells, which are required for the structural integrity and filtration function of capillary tufts; knockout studies in model organisms have demonstrated that loss of PDGFRB or its ligand PDGF-B leads to defective glomerular morphogenesis and vascular abnormalities (betsholtz2001developmentalrolesof pages 11-13). In addition to its developmental roles, PDGFRB is involved in tissue repair processes; for instance, PDGF-mediated signaling is pivotal for wound healing by stimulating fibroblast proliferation, migration and the synthesis of extracellular matrix components (hollinger2008recombinanthumanplateletderived pages 1-2, donovan2013plateletderivedgrowthfactor pages 1-3). Furthermore, aberrant activation of PDGFRB has been implicated in various pathological conditions including fibrotic diseases, atherosclerosis, and oncogenesis; in many tumors, PDGFRB is expressed in stromal cells and pericytes, where it supports neovascularization, tumor cell survival, and the establishment of a pro-tumorigenic microenvironment (raica2010plateletderivedgrowthfactor pages 14-16, rosenkranz1999evidencefordistinct pages 5-6). Downstream signaling of PDGFRB engages multiple pathways such as the PI3K-Akt pathway, the Ras-MAPK cascade, and PLC-γ-mediated calcium signaling, which collectively regulate transcriptional programs associated with cell cycle progression and cytoskeletal rearrangements (kazlauskas1992gtpaseactivatingproteinand pages 1-2, valius1993tyrosines1021and pages 9-10).
8. Other Comments  
   PDGFRB is a notable target in clinical oncology and anti-fibrotic therapy. Specific tyrosine kinase inhibitors, such as imatinib, have proven effective in treating neoplasms driven by constitutively active PDGFRB fusion proteins; such fusion proteins, commonly observed in certain myeloproliferative disorders, result from chromosomal rearrangements that remove normal regulatory domains and lead to ligand-independent activation (pierce1995detectionofplateletderived pages 14-15, maudsley2000plateletderivedgrowthfactor pages 10-11). In addition, neutralizing antibodies against PDGFRB have been developed to block ligand binding and receptor activation, thereby reducing fibroblast proliferation and collagen deposition in fibrotic diseases (gilbertson2001plateletderivedgrowthfactor pages 1-2). PDGFRB is also implicated in vascular pathologies; its overactivation in vascular smooth muscle cells contributes to neointima formation and restenosis following arterial injury (magnusson2007plateletderivedgrowthfactor pages 16-19). Ongoing research focuses on the development of more specific inhibitors and combination therapies that target PDGFRB alongside related angiogenic pathways, given the receptor’s central role in coordinating pericyte recruitment and vessel stabilization in both physiological and pathological states (raica2010plateletderivedgrowthfactor pages 21-23, raica2010plateletderivedgrowthfactor pages 23-26). The potential for cross-talk with other receptors, including PDGFR alpha and vascular endothelial growth factor receptors (VEGFRs), further underscores the complexity of PDGFRB’s regulatory network and its importance as a therapeutic target (rosenkranz1999evidencefordistinct pages 7-8).
9. References  
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   gilbertson2001plateletderivedgrowthfactor pages 6-7  
   magnusson2007plateletderivedgrowthfactor pages 16-19  
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   rosenkranz1999evidencefordistinct pages 5-6  
   rosenkranz1999evidencefordistinct pages 7-8

This nomenclature and functional profile of PDGFRB strictly reflects data derived from peer‑reviewed publications, providing a detailed overview of its phylogeny, catalytic reaction, cofactor requirements, substrate specificity, three‑dimensional structure, regulatory mechanisms, biological functions and additional clinical or research‑related insights without interpretation of the experimental findings.

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