## 1. Phylogeny

Platelet‐derived growth factor receptor alpha (PDGFRA) is a receptor tyrosine kinase that belongs to the type III receptor tyrosine kinase family, which also includes receptors such as PDGFRβ, KIT, CSF1R, and FLT3. (guerit2021pdgfreceptormutations pages 1-2) Orthologs of PDGFRA are found across all vertebrate species, underscoring its highly conserved role in the regulation of fundamental cellular processes during evolution. (farahani2015plateletderivedgrowthfactor pages 1-2) Comparative genomic analyses indicate that the evolutionary origins of PDGFRA can be traced back to early eukaryotic ancestors, with conservation of key structural domains that mediate ligand recognition and catalytic function. (nazarenko2012pdgfandpdgf pages 1-3) As part of the broader kinome, PDGFRA shares significant sequence and functional homology with other receptor tyrosine kinases within the type III subfamily, which have evolved to collectively regulate cell proliferation, differentiation, and survival across a wide phylogenetic spectrum. (guerit2021pdgfreceptormutations pages 1-2)

## 2. Reaction Catalyzed

PDGFRA functions as a receptor tyrosine kinase that, upon activation, catalyzes the transfer of a phosphate group from ATP to specific tyrosine residues on substrate proteins. (heinrich2003pdgfraactivatingmutations pages 2-3) The chemical reaction it mediates can be summarized as follows: ATP + [protein]–tyrosine → ADP + [protein]–phosphotyrosine + H⁺; this reaction represents the fundamental biochemical event underlying PDGFRA signal transduction. (guerit2021pdgfreceptormutations pages 2-5) Autophosphorylation of tyrosine residues within the kinase domain further amplifies receptor activation and creates docking sites for downstream adaptor proteins, thereby initiating multiple signaling cascades. (mcdermott2009liganddependentplateletderivedgrowth pages 1-2)

## 3. Cofactor Requirements

The catalytic activity of PDGFRA is dependent on the presence of ATP and requires divalent metal ions, with magnesium (Mg²⁺) being the primary cofactor that coordinates ATP binding and facilitates the phosphoryl transfer reaction. (guerit2021pdgfreceptormutations pages 14-15) As observed with other receptor tyrosine kinases, the binding of Mg²⁺ stabilizes the transition state and is essential for efficient catalysis during receptor activation. (mcdermott2009liganddependentplateletderivedgrowth pages 1-1, heinrich2003pdgfraactivatingmutations pages 1-2)

## 4. Substrate Specificity

PDGFRA displays substrate specificity that is characteristic of receptor tyrosine kinases, preferentially phosphorylating tyrosine residues on both itself (through autophosphorylation) and on select downstream targets. (mcdermott2009liganddependentplateletderivedgrowth pages 9-10) Although a detailed consensus phosphorylation motif for PDGFRA substrates is not explicitly defined in the current literature provided, the receptor’s activity leads to the phosphorylation of specific tyrosine residues that create binding sites for SH2 domain–containing proteins, thereby regulating the recruitment of downstream signaling complexes. (guerit2021pdgfreceptormutations pages 2-5, heinrich2003pdgfraactivatingmutations pages 2-3)

## 5. Structure

PDGFRA is organized into several distinct structural domains that collectively mediate ligand-binding, dimerization, and catalytic activity. The N-terminal extracellular region comprises five immunoglobulin-like (Ig-like) domains responsible for binding PDGF ligands such as PDGFA, PDGFB, and PDGFC. (nazarenko2012pdgfandpdgf pages 3-4) This ligand-binding domain is followed by a single transmembrane helix that anchors the receptor in the plasma membrane and plays a critical role in receptor dimerization upon ligand engagement. (mcdermott2009liganddependentplateletderivedgrowth pages 3-4) Immediately adjacent to the transmembrane segment is the juxtamembrane region, which functions in autoinhibition by masking the kinase domain in the inactive state and regulating the transition to an active conformation. (farahani2015plateletderivedgrowthfactor pages 7-8) The intracellular portion of PDGFRA contains a highly conserved tyrosine kinase domain, which features an ATP-binding pocket, an activation loop, and catalytic motifs critical for phosphoryl transfer. Key structural elements such as the C-helix and hydrophobic spines are essential for the stabilization of the active conformation following receptor dimerization and autophosphorylation. (heinrich2003pdgfraactivatingmutations pages 2-3, nazarenko2012pdgfandpdgf pages 3-4) In addition, computational and experimental analyses of the transmembrane domain have revealed that specific mutations, for example in the V536E variant, can alter helix packing and promote constitutive receptor activation by favoring an active dimeric configuration. (polyansky2019atomisticmechanismof pages 2-3)

## 6. Regulation

Regulation of PDGFRA activity is achieved chiefly by ligand binding, which induces receptor dimerization and activates the intrinsic kinase activity via autophosphorylation of discrete tyrosine residues. (mcdermott2009liganddependentplateletderivedgrowth pages 1-2, heinrich2003pdgfraactivatingmutations pages 2-3) In its inactive state, the juxtamembrane domain exerts an autoinhibitory influence on the kinase domain, preventing spontaneous activation; ligand-induced conformational rearrangements relieve this inhibition and enable a transition to an active state. (guerit2021pdgfreceptormutations pages 2-5) Post-translational modifications, most notably phosphorylation, serve as critical regulatory switches that not only promote kinase activity but also create binding sites for downstream signaling proteins, thereby propagating intracellular signals. (mcdermott2009liganddependentplateletderivedgrowth pages 9-10) In addition, regulatory mechanisms include microRNA-mediated suppression; for instance, miR-146b has been shown to bind the 3′ untranslated region of PDGFRA mRNA, reducing its expression and consequently affecting pathways involved in erythropoiesis and megakaryocytopoiesis. (zhai2014theregulatoryroles pages 1-2, zhai2014theregulatoryroles pages 3-6) Mutations in PDGFRA, such as those affecting the activation loop (e.g., D842V) or transmembrane domain, disrupt normal regulatory processes and lead to ligand-independent, constitutive activation that is frequently observed in various human cancers. (heinrich2003pdgfraactivatingmutations pages 2-3, guerit2021pdgfreceptormutations pages 2-5) Furthermore, pharmacological inhibitors—including imatinib, sunitinib, and crenolanib—can block receptor phosphorylation by stabilizing inactive conformations, thereby providing a means to therapeutically modulate PDGFRA activity. (guerit2021pdgfreceptormutations pages 14-15, paugh2013noveloncogenicpdgfra pages 11-13)

## 7. Function

PDGFRA serves as a critical mediator of cell-surface signaling by binding platelet-derived growth factor ligands, notably PDGFA, PDGFB, and PDGFC, to regulate extensive biological processes. (OpenTargets Search: -PDGFRA, farahani2015plateletderivedgrowthfactor pages 7-8) In the context of embryonic development, PDGFRA is essential for normal skeletal formation, cephalic closure, and the development of the gastrointestinal tract mucosa, where it facilitates the recruitment of mesenchymal cells and contributes to the formation of intestinal villi. (farahani2015plateletderivedgrowthfactor pages 7-8) Beyond development, PDGFRA plays pivotal roles in cell proliferation, survival, and migration; its activation can either promote or inhibit cell division and motility depending on the cellular and tissue context. (guerit2021pdgfreceptormutations pages 1-2) In bone marrow–derived mesenchymal stem cells, PDGFRA is critically involved in the differentiation process, thereby influencing the formation of bone, cartilage, and adipose tissue. (OpenTargets Search: -PDGFRA, farahani2015plateletderivedgrowthfactor pages 7-8) In addition, PDGFRA-mediated signaling is important for wound healing, as it regulates chemotaxis and extracellular matrix deposition, and it can participate in platelet activation and aggregation through its effects on downstream effectors. (mcdermott2009liganddependentplateletderivedgrowth pages 1-2) Dysregulation of PDGFRA—often by way of activating mutations or overexpression—has been strongly linked to oncogenic processes in a variety of cancers, including gastrointestinal stromal tumors (GISTs), certain gliomas, and other malignancies where aberrant signal transduction leads to uncontrolled cell proliferation and enhanced survival. (guerit2021pdgfreceptormutations pages 1-2, li2022thepdgffamily pages 6-7, mcdermott2009liganddependentplateletderivedgrowth pages 9-10)

## 8. Other Comments

Numerous small molecule inhibitors have been developed targeting PDGFRA, reflecting its importance as a therapeutic target. Imatinib mesylate was one of the first drugs used in clinical settings to inhibit PDGFRA activity in gastrointestinal stromal tumors; however, mutations such as D842V have been associated with imatinib resistance, necessitating the development of alternative inhibitors like crenolanib and dasatinib. (heinrich2003pdgfraactivatingmutations pages 2-3, guerit2021pdgfreceptormutations pages 14-15, paugh2013noveloncogenicpdgfra pages 11-13) In addition, recent drug repurposing efforts have explored compounds such as itraconazole for their potential anti-neoplastic effects via PDGFRA inhibition, with in silico docking studies supporting strong binding affinities that compare favorably with established kinase inhibitors. (arulanandam2021repurposingofan pages 12-15) Germline mutations in PDGFRA have been identified in familial syndromes characterized by the development of multiple gastrointestinal mesenchymal tumors, including inflammatory fibroid polyps and GISTs. Such mutations, exemplified by amino acid substitutions like P653L, further highlight the clinical relevance of PDGFRA in tumor predisposition syndromes. (ricci2015pdgframutantsyndrome pages 6-7, ricci2015pdgframutantsyndrome pages 7-8, ricci2015pdgframutantsyndrome pages 8-9) Structural investigations of the PDGFRA transmembrane domain, including atomistic modeling of mutations such as V536E, have uncovered mechanistic insights into how altered helix packing can lead to constitutive receptor dimerization and activation, emphasizing novel opportunities for targeted intervention. (polyansky2019atomisticmechanismof pages 11-12) Moreover, PDGFRA expression within the tumor stroma has been correlated with increased extracellular matrix deposition, elevated matrix stiffness, and poor clinical prognosis in cancers such as ovarian carcinoma and in the stromal compartment of the mammary gland, thereby linking receptor signaling not only to tumor cell autonomous growth but also to the modulation of the tumor microenvironment. (hammer2017stromalpdgfrαactivation pages 3-4, li2022thepdgffamily pages 6-7) Together, these advancements underscore the multifaceted roles of PDGFRA in both normal physiology and disease, while also highlighting its value as a target for therapeutic modulation in diverse clinical contexts.

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