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Review Article

Antagonists of platelet-derived growth factors (PDGFs) and their receptors (PDGFRs) as rationale and therapeutic potential of combination cancer therapy

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Abstract: Platelet-Derived Growth Factor (PDGF)/Platelet-Derived Growth Factor Receptor (PDGFR) are key regulators in the complex network of signaling from Receptor Tyrosine Kinases (RTKs). PDGFR has historically been considered an attractive therapeutic target given its association with oncogenic mutations, gene fusions and altered signaling. However, growing evidence suggests that PDGFR functions beyond these traditional roles as a single therapeutic target and into a broader role as a central coordinator of oncogenic signaling. PDGFR mediates cross-talk among multiple oncogenic pathways such as EGFR, VEGFR and IGF-1R thus supporting redundancy in oncogenic signaling, resistance to therapy and adaptive changes in tumors. The biologic effects of PDGFR signaling do not end at tumor cell borders but rather include the regulation of the tumor microenvironment. Specifically, PDGFR directs the recruitment of pericytes, remodeling of the extracellular matrix and influences interstitial fluid pressure (IFP); each effect ultimately impacts both drug delivery and therapeutic efficacy. Studies have demonstrated that inhibition of PDGFR reduces stromal tension and normalizes tumor vasculature resulting in enhanced penetration of chemotherapy, targeted and radiation therapy. As such, these studies provide rationale for using PDGFR directed strategies in order to enhance treatment outcome via rational combinations of drugs. This review aims to integrate current knowledge of PDGF/PDGFR biology based on molecular interactions, tumor microenvironment dynamics and therapeutic approaches. It will highlight how PDGFR directed therapeutic strategies may serve to circumvent drug resistance, enhance drug delivery, enable use of biomarkers to drive combination therapeutics and promote a systems-level understanding of PDGFR function as a basis for advancing its translational application in precision oncology.

Keywords: PDGF/PDGFR, co-expression, cross-talk, drug delivery, cancer treatments, combination therapies

Introduction

The platelet derived growth factor (PDGF)/platelet derived growth factor receptor (PDGFR) pathway is a major hub for signal transduction through receptor tyrosine kinases (RTK), controlling tumor cell proliferation, interaction with the stroma and remodeling of blood vessels. Mutations, amplification or fusions of the PDGFR genes are among the most frequent alterations found in various types of tumors including hematologic cancers. Therefore, the PDGFR is considered a versatile therapeutic target in cancer treatment [1, 2]. Furthermore, since PDGFR has been implicated in numerous other pathways such as angiogenesis, inflammation and apoptosis, it plays more of a system level regulatory function rather than simply being a single oncogene to drive tumor progression. This integrated view is also reflected by recent studies aiming at developing new biomarkers for therapy decision making and rational combinations of treatments.

PDGFR proteins undergo diverse oncogenic alterations, which include changes from genetic mutation at all three parts of the protein, including extracellular, transmembrane and

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Table 1. PDGFR-targeting agents

Agent	Type	Target	Mechanism	Clinical Context
Imatinib	TKI	PDGFRβ	Inhibits kinase activity, reduces IFP	GIST, NSCLC
IB3	Antibody	PDGFRβ	Blocks ligand binding	Preclinical (pancreatic)
Aptamer (PDGFB)	Nucleic acid	PDGF-B	Ligand neutralization	Experimental
Pazopanib	Multi-TKI	VEGFR/PDGFR	Anti-angiogenic	Solid tumors

kinase, and changes due to extensive fusion events between genes, especially in blood cancers [1, 3]. The changes that result from these mutations cause the abnormal activation of downstream signaling pathways and demonstrate why PDGFR can act as both a potential biomarker for diagnosis and an additional therapeutic target. Further, PDGFR's signaling pathway has been found to interact with several other oncogenetic pathways because of their co-expression patterns and cross-talk mechanisms, demonstrating how PDGFR plays a key role in coordinating the development of tumors [4, 5]. Further, the PDGFs and their receptors are involved in cross-talks with cell-surface receptor proteins such as AXL protein receptors and G-protein coupled receptors (GPCR) implicated as oncogenes and other RTKs, and their inhibition has proven to facilitate the delivery of various therapeutic agents. These essential characteristics could be harnessed to design combination therapies in cancer treatments [2, 4-10]. Representative PDGFR-targeting agents and their mechanisms are summarized in **Table 1**.

One major obstacle to delivering chemotherapy drugs to tumors involves overcoming barriers that limit their ability to penetrate tumor tissue. The PDGFR signaling pathway helps create barriers to drug delivery through its effects on the architectural arrangement of stromal cells, vessel integrity, and interstitial fluid pressure, all factors that affect how well drugs can reach the target site [11, 12]. Studies have also demonstrated that inhibiting PDGFR can lead to reduced stromal stiffness and enhanced vascular function, leading to improved delivery and effectiveness of both chemotherapeutic and targeted therapies [13, 14].

Beyond its signal transduction roles, PDGFR has potential as a marker or diagnostically useful molecule. As part of certain gene fusion events that can be tumor specific to some degree, PDGFR offers opportunities for targeted biomarker discovery and therapy [15]. Biomarkers associated with PDGFR may also provide evidence about how patients will respond to treatments, thereby facilitating the use of precision oncology methods in the clinic [16, 17].

In summary, the integrated nature of PDGF/PDGFR's multiple functional roles in promoting oncogenic signal transduction, regulating tumor microenvironments, and delivering drugs therapeutically underscores the potential of targeting PDGF/PDGFR as a key therapeutic target for cancer. As such, this paper provides an integrated overview of the PDGF/PDGFR pathway, with emphasis on its function within cellular signaling pathways, drug-resistant pathways, and drug delivery systems. By examining the integration of all three areas, we demonstrate how PDGFR-targeted treatments could lead to rationally designed combination therapies that improve patient outcomes.

PDGFs and PDGFRs involvement in networks of molecular interactions with other oncogenes

PDGFR signaling, rather than acting independently, often interact with several other oncogenic pathways to help the tumor behave. A similar sequence of events is found among many different kinds of cancer that involve PDGFR activation along with VEGF and/or HIF-1α promoting angiogenesis, and with EGFR and/or FGFR signaling to promote proliferation. In addition to being cooperative, these signals have been shown to be functionally synergistic, providing tumors with new capabilities like drug-resistance and protection from their micro-environment. The convergence of PDGFR signaling into a central role in coordinating support systems for the tumor, provides a rationale for developing multi-component therapeutic approaches designed to disrupt one or more of these tumor-supportive processes concurrently [2, 5, 18, 19].

PDGF receptor interaction encompasses a variety of non-classical ligand-receptor signal transduction processes such as gene fusion, extracellular matrix (ECM) interactions and post-translational regulation. Collectively, these provide an increased likelihood for aberrant pathway activation contributing to continued tumor growth via modification of both the intensity and duration of signaling [1, 20]. Although these molecular interactions represent a broad range of possibilities, they all lead to the activation of common downstream pathways, namely PI3K-AKT, RAS-ERK, and NF-kappa B signaling, that together facilitate tumor cell survival, proliferation, and inflammation [21].

The functional implications associated with the multitude of interconnected PDGFR signaling events are best understood and appreciated by evaluating them within the framework of specific tumor phenotypes.

Functional integration of PDGFR signaling in cancer phenotypes

The significance of PDGFR coexpression and cross-talk becomes clear when viewed from the perspective of tumor phenotype, in which signaling interactions produce biologically relevant consequences for disease development. Instead of functioning as an isolated receptor, PDGFR signaling mediates several major oncogenically important processes in tumors, that is, tumor microenvironment remodeling, drug resistance, and angiogenesis with metastatic capacity [2, 21].

Stromal fibroblast and pericyte activation mediated by PDGFR are critical in tumor microenvironment remodeling through the enhancement of extracellular matrix deposition and increases in interstitial fluid pressure (IFP), resulting in a physical and biochemical barrier that hinders drug diffusion into tumor tissues [11-13]. Evidence has shown that experimental inhibition of PDGFR signaling decreases IFP, thereby facilitating greater delivery and efficacy of chemotherapy agents, emphasizing the impact of PDGFR on both mechanical properties of tumor tissues and access to drugs [13, 14].

Finally, PDGFR signaling plays a crucial role in promoting angiogenesis and metastasis through interaction with vascular and hypoxia related pathways. Its coordination with VEGF and HIF-1α promotes the maturation and stability of blood vessels that support long-term tumor growth and dissemination [18, 19, 22]. While this angiogenic function may increase tumor perfusion it creates a paradox whereby PDGFR activity simultaneously supports tumor progression while enhancing drug delivery thus requiring context-dependent therapeutic approaches [23].

Aside from its effects on stromal regulation, PDGFR interacts with other RTK's such as EGFR and VEGFR, producing redundant survival signaling networks contributing to resistance against therapy. The convergent action of these receptors on common downstream pathways including PI3K-AKT and MAPK signaling cascades allows tumor cells to maintain their proliferative capacity and survive despite targeted inhibitors [5, 24]. A clinical example of this is co-activation of PDGFR and EGFR having been linked to poor response to EGFR-targeting therapy in bladder carcinoma patients demonstrating the clinical relevance of signaling redundancy in resistance mechanisms [25].

These mechanisms illustrate that PDGFR is not simply another receptor that happens to be expressed together with others but rather functions as an integrator of cellular, stromal and vascular network functions that link them all. As such its integrative function provides justification for targeting PDGFR as part of therapeutic regimens to overcome resistance and improve treatment efficacy especially when using combination strategies.

Key PDGFR interactions and their therapeutic implications across cancer types are summarized in **Table 2**.

PDGF/PDGFR complex targeting facilitates drug delivery in cancer

Drug delivery in oncology is limited by physiological and pathological barriers in solid tumors including abnormal blood vessel formation and increased interstitial fluid pressure. As a result, PDGFR signaling can influence these structural elements and limit delivery of therapeutic agents into the tumor site. Therefore, modulating the tumor micro environment via PDGFR inhibition provides a mechanistically based method to improve drug delivery. Representative PDGFR-targeting agents, their me-

Table 2. PDGFR interactions and combination strategies

Cancer Type	Co-Pathway	Mechanism	Therapeutic Strategy
NSCLC	EGFR	Resistance via cross-talk	EGFR + PDGFR inhibition
Glioblastoma	VEGF/HIF-1α	Angiogenesis	Anti-VEGF + PDGFR
Pancreatic	Stromal PDGFRβ	Drug penetration barrier	PDGFR inhibitor + chemo
Colorectal	mTOR/PDGF	Metastasis	Dual pathway blockade

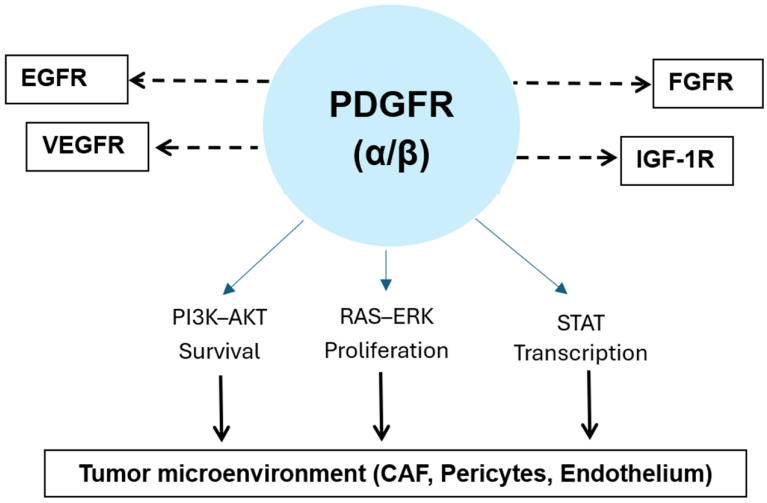

Figure 1. PDGFR-centered signaling network and cross-talk with oncogenic receptor tyrosine kinases. PDGFR (α/β) interacts with EGFR, VEGFR, FGFR, and IGF-1R through cross-talk mechanisms (dashed arrows). Activation of PDGFR triggers key downstream pathways, including PI3K-AKT (survival), RAS-ERK (proliferation), and STAT (transcription), contributing to tumor progression and therapeutic resistance within the tumor microenvironment.

A schematic representation of PDGFR-centered signaling integration and cross-talk with oncogenic pathways is shown in **Figure 1**.

The effects of PDGFR inhibition on interstitial fluid pressure, vascular normalization, and improved drug delivery are illustrated in **Figure 2**.

Collectively, these findings demonstrate that PDGFR targeting is an effective mechanism to enhance the delivery of therapeutic agents into solid tumors either directly or indirectly through alteration of tumor physiology, thereby, reinforcing its essence in the design of combination therapy.

chanisms, and therapeutic applications are summarized in **Table 1**.

PDGFR inhibition has demonstrated the ability to lower IFP and to normalize the vascular structure in addition to lowering stromal resistance which facilitates greater uptake of chemotherapeutic agents, targeted therapies, and radiotherapeutic agents. Pharmacologic inhibitors of PDGFR have lowered stromal resistance facilitating improved delivery of chemotherapeutic agents such as docetaxel [14] and liposomal doxorubicin [13], while antibodies and peptides have been used as selective targeting molecules to deliver therapeutics to tumor and stromal cells expressing PDGFR, resulting in higher intracellular concentrations of delivered agents [26, 27]. The effects of PDGFR on normalizing the tumor's blood flow has also improved the delivery of radioactive isotopes for use in radiotherapy. Improved perfusion and a reduced tumor to normal tissue ratio were observed when using PDGFR inhibitors with radiolabeled agents [28, 29].

Targeted PDGFR-mediated drug delivery mechanisms are illustrated in **Figure 3**.

PDGFR as a strategic target in combination cancer therapies

As the complexity of biological systems involved in cancer evolves, and given that cancer is now recognized as being capable of adapting to new forms of therapy, and capable of creating a protective environment around itself based on interactions with its microenvironment, it is increasingly necessary to move away from monotherapy approaches, and toward rationally designed combinations of therapies. In this light, the PDGF/PDGFR axis represents an especially promising target for multi-modal therapies since PDGFR serves as both a regulatory node in signaling pathways controlling the proliferation of tumor cells, and a system-wide controller of structural and functional characteristics of the tumor micro-environment.

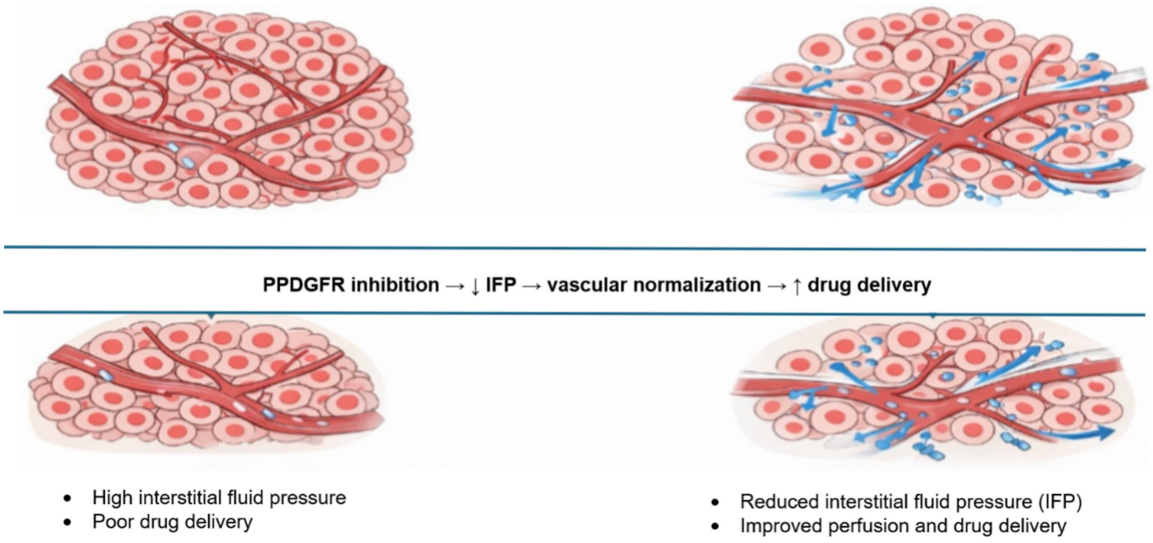

Figure 2. PDGFR inhibition remodels the tumor microenvironment to enhance drug delivery. Tumors with high interstitial fluid pressure (IFP) exhibit poor perfusion and limited drug penetration. Inhibition of PDGFR reduces IFP, promotes vascular normalization, and improves perfusion, resulting in enhanced delivery of therapeutic agents.

Functionally, PDGFR promotes resistance through its participation in RTK networks, where PDGFR interacts with other receptors and pathways, including EGFR, VEGFR, and FGFR to generate common downstream effectors, for instance, PI3K-AKT/MAPK that maintain proliferative/survival signals even when one or more of these pathways are inhibited [5, 24]. Signaling convergence among tumor cells enables them to bypass therapeutic inhibition of specific pathways and continue to receive growth/survival signals. Therefore, simultaneous targeting of PDGFR and these convergent pathways disrupts redundant compensation signaling loops, reducing the redundancy in pathways and enhancing the efficacy of single-agent targeted therapies (**Table 2**).

Concurrently, PDGFR is a major contributor to establishing the structure/function of the tumor micro-environment, specifically through its influence on stromal cells, ECM composition and vascular organization. All three factors significantly impact interstitial fluid pressure (IFP), which is a key determinant of drug delivery into solid tumors. Reducing stromal tension through PDGFR inhibition enhances vascular function and facilitates greater delivery of therapeutic agents into solid tumors [11-13]. **Figure 2** illustrates how PDGFR inhibition reduces IIFP, resulting in better delivery of therapeutics into solid tumors.

Moreover, PDGFR directed combination strategies contribute to chemosensitizing, where PDGFR inhibition removes stromal barriers to the delivery of chemotherapy, making tumors more susceptible to subsequent therapies. For instance, studies have demonstrated that PDGFR inhibition improves the uptake/efficacy of various cytotoxics and radio-labeled antibodies by improving blood flow within solid tumors and lowering the resistance generated by the stroma [14, 29]. These therapeutic modalities include targeted inhibitors, antibodies and ligand-directed delivery systems are summarized in **Table 1**, with illustration of receptor mediated targeting mechanisms in **Figure 3**. Additionally, consideration of the complex nature of PDGF ligands' functions, including context-dependent effects on angiogenesis and metastasis, highlights the necessity of designing combination therapies carefully, and identifying appropriate combination partners [23].

Therefore, the translational value in combination therapy using PDGFR as a strategic target will rely on the simultaneous addressing of all aspects contributing to failure in current treatments: redundancy in signaling; barrier created by the micro-environment to resist therapies; poor delivery of therapeutic agents into solid tumors. Ultimately future progress will be made possible by combining mechanistic knowledge with biomarker-based patient selection

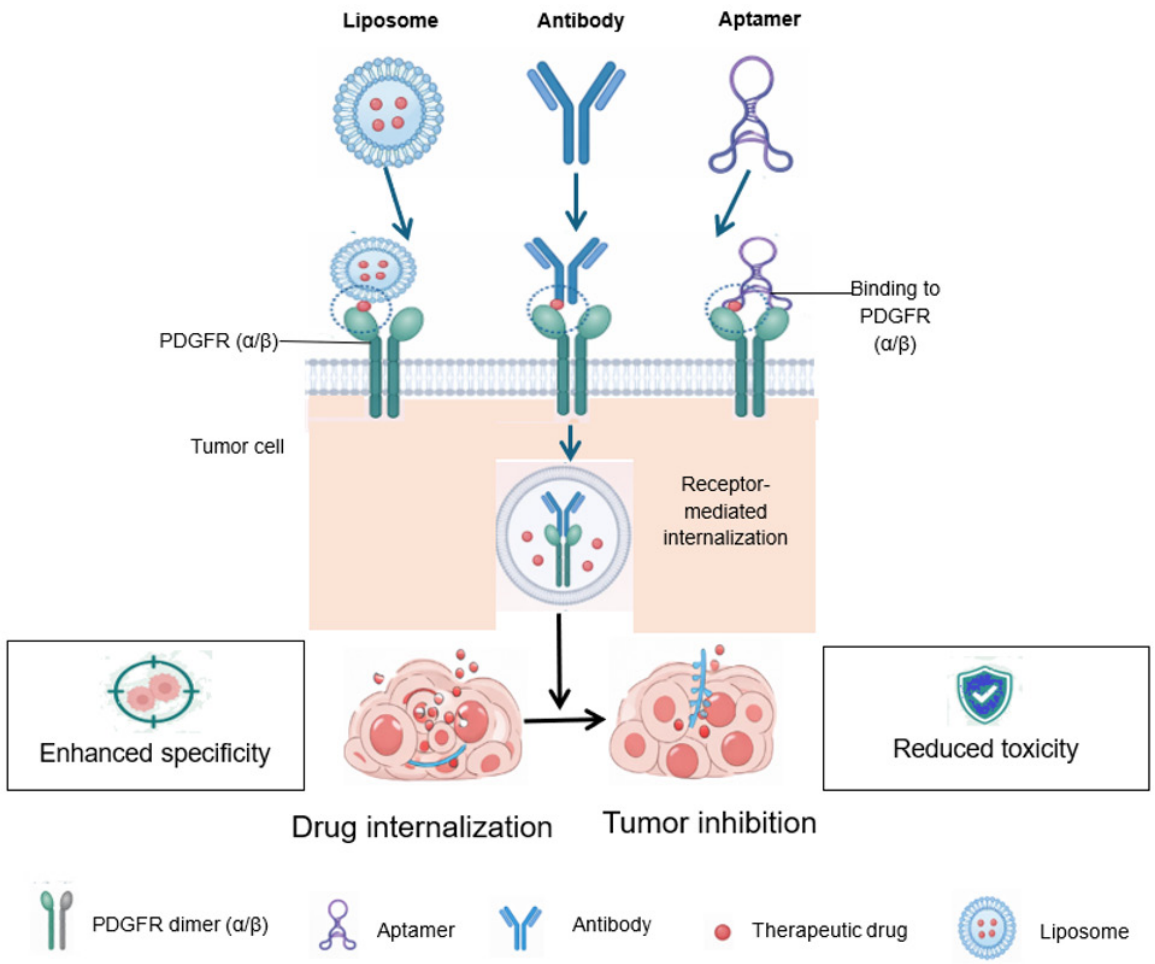

Figure 3. PDGFR-targeted drug delivery and receptor-mediated internalization. Liposomes, antibodies, and aptamers selectively bind to PDGFR (α/β) expressed on tumor and stromal cells, facilitating receptor-mediated internalization and intracellular drug accumulation. This targeted approach enhances therapeutic specificity while reducing systemic toxicity.

criteria to identify patients who would most benefit from interventions involving PDGFR.

In this context, the PDGF/PDGFR axis should be viewed as part of a larger systems-level control mechanism that regulates the biology of tumors, rather than simply another singular therapeutic target, and modulating this axis as a means to reorganize tumor biology and improve the effectiveness of multifaceted cancer therapies.

Conclusion

The PDGF/PDGFR signaling pathway is at the center of the multiple interacting systems involved in tumor development and progression, as well as stroma and vessel formation. This receptor tyrosine kinase does not act solely as a conventional receptor tyrosine kinase, but rather it acts as an interface or coordinator of tumor-stromal interaction and adaptive resistance.

Studies both in vitro and in vivo have shown that PDGFR targeted therapy provides short-term results due to cross-talk with other pathways while being a good target for combination therapy due to its involvement in oncogenetic networks. Therefore, using a combination of PDGFR inhibitors and anti-angiogenic, pro-apoptotic and immunotherapeutic drugs could provide a rational strategy to prevent resistance and improve the effectiveness of treatments.

However, future advances will be dependent upon developing validated biomarkers able to identify which patient populations are likely to

receive maximum benefits from PDGFR targeted therapy. Also, further research is necessary to develop a better understanding of how different cell types respond to PDGFR activation in the context of the tumor microenvironment to optimize the timing, sequence and selection of drugs used in combination.

Therefore, the clinical utility of PDGFR directed therapies is related to their inclusion into multimodal treatment protocols that are specifically designed to inhibit the integrated biology of cancers.

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Disclosure of conflict of interest

None.

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