gossamerbio®

Corporate Presentation

September 2026

Forward Looking Statements

This Presentation contains forward-looking statements. All statements other than statements of historical facts contained in this presentation, including statements regarding: the Company's interpretation of the FDA minutes from the Company's Pre-NDA Type B meeting with the FDA; the timing and potential submission, and potential acceptance for filing and approval, of an NDA for seralutinib in PAH; the potential significance, interpretation and implications of data from the Phase 3 PROSERA study and Phase 2 TORREY study and supportive analyses; the development potential and market opportunity of seralutinib in PAH, PH-ILD and other indications; and our future business strategies and plans, research and development plans, and the timing and likelihood of success, plans and objectives of management for future operations. In some cases, you can identify forward-looking statements by terms such as "may," "will," "should," "expect," "plan," "anticipate," "could," "intend," "target," "project," "contemplates," "believes," "estimates," "predicts," "potential" or "continue" or the negative of these terms or other similar expressions.

The inclusion of forward-looking statements should not be regarded as a representation by us that any of our plans will be achieved. Actual results may differ from those set forth in this presentation due to the risks and uncertainties inherent in our business, including, without limitation: the risk that the Company's planned NDA submission is based in part on its views following its recent meeting with the FDA and the official minutes therefrom and later feedback from the FDA, which may be inconsistent with such meeting or the Company's views from such meeting; later developments with the FDA may be inconsistent with the feedback from prior meetings; the FDA may determine that our planned NDA does not qualify for filing; the results of the Company's clinical trials, including the Phase 3 PROSERA and Phase 2 TORREY studies, may not be deemed sufficient by the FDA to serve as the basis for regulatory approval of seralutinib, including the risk that the FDA determines that the overall benefit-risk assessment of seralutinib is not favorable; or may limit the commercial opportunity for seralutinib; our future performance is dependent entirely on the success of seralutinib; the Company's interpretation, significance and regulatory relevance of data from the Phase 3 PROSERA study, including the CT FRI substudy, may be inconsistent with the views of the FDA or others; whether the anticipated benefits of the Company's new capital structure are realized; potential delays in the commencement, enrollment and completion of clinical trials; the Company's dependence on third parties in connection with product manufacturing, research and preclinical and clinical testing; the results of preclinical studies and early clinical trials with seralutinib are not necessarily predictive of future results; the success of Gossamer's clinical trials and preclinical studies for seralutinib; regulatory developments in the United States and foreign countries; adverse side effects or inadequate efficacy of seralutinib that may limit its development, regulatory approval and/or commercialization, or may result in clinical holds, recalls or product liability claims; Gossamer's ability to obtain and maintain intellectual property protection for seralutinib; Gossamer may use its capital resources sooner than it expects; and other risks described in the Company's prior press releases and the Company's filings with the Securities and Exchange Commission ("SEC"), including under the heading "Risk Factors" in the Company's annual report on Form 10-K and any subsequent filings with the SEC. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof, and we undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date hereof. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995.

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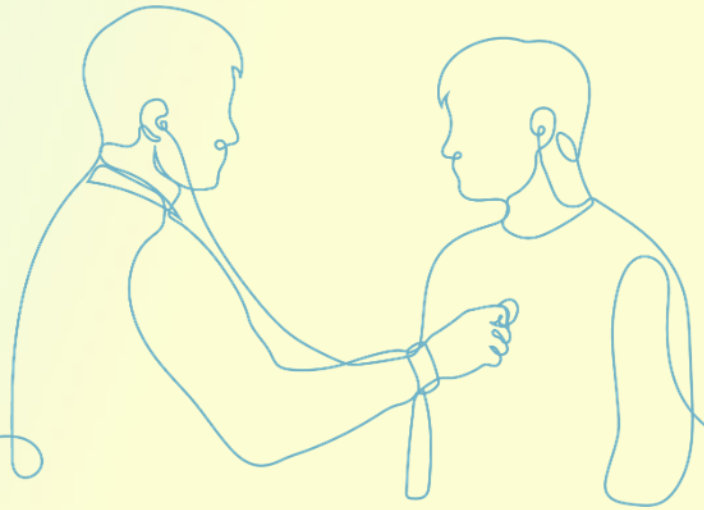
I. Overview



Gossamer Bio Overview

HQ in San Diego, California

Focused on developing treatments for fatal rare lung diseases

Worldwide Rights to Seralutinib

Comp. of Matter IP Patent Protection until 2039(1)

PROGRAM	CLASS (Route of Admin.)	INDICATION	PHASE 1	PHASE 2	PHASE 3	NDA Filed	Approved
Seralutinib (GB002)	PDGFR, CSF1R, c-KIT Inhibitor (Inhaled)	Pulmonary Arterial Hypertension (PAH)	Completed Phase 3 PROSERA Study in PAH Completed Type B Pre-NDA Meeting with FDA			**Expected NDA Submission**	
		Pulmonary Hypertension Associated with Interstitial Lung Disease (PH-ILD)	Future Development: Phase 3 Ready				

(1): Includes assumption of maximum five years of patent term extension. Corresponding patent(s) and patent application(s) with compound-specific claims. Total patent life with patent term extension cannot exceed 14 years from approval.

Potential First-in-Class, Inhaled TKI for the Treatment of Pulmonary Hypertension

Seralutinib is a potential first-in-class inhaled TKI, a mechanism and therapeutic class distinct from all current PAH therapies[1]

Focused on addressing the underlying disease pathophysiology of PAH, targeting PDGFR, CSF1R, and c-KIT[1]

Purpose built for PAH in wake of Phase 3 IMPRES (imatinib) findings[1,2,3]

- Designed to be more potent and selective for kinases relevant in PAH, while avoiding c-abl[1]
- Inhaled delivery is designed to increase drug exposure at the site of disease while mitigating systemic AEs[1,2,3]

(1): Galkin A et al. Eur Respir J. 2022;60:2102356. (2): Hoeper MM et al. Circulation. 2013;127:1128-1138. (3): Frost AE et al. J Heart Lung Transplant. 2015;34:1366-1375. TKI = tyrosine kinase inhibitor; PDGFR = platelet-derived growth factor receptor; CSF1R = colony stimulating factor 1 receptor; c-KIT = mast/stem cell growth factor receptor (KIT); c-Abl = Abelson tyrosine kinase; BMPR2 = bone morphogenetic protein receptor type 2; PH = pulmonary hypertension; PAH = pulmonary arterial hypertension; AEs = adverse events.

Seralutinib Offers A Strong Value Proposition

Near-Term Value Inflection

- Completed Phase 3 in a heavily pre-treated PAH population(1)
- Clinically meaningful, statistically supported gains on primary and key secondary endpoints that were amplified in higher-risk patients(2,3)
- Well tolerated
- Planned NDA submission and potential FDA decision 3Q27(4)

First-in-Class Potential with Evidence of Disease Remodeling

- Complementary mechanism targeting the pathophysiology of the disease: anti-remodeling agent(5)
- Durable, deepening effect through Week 72 in Phase 2 OLE (PVR) and Week 48 in Phase 3 (6MWD) – biologically consistent with MoA(2,5,6)
- PROSERA CT FRI substudy, the most comprehensive controlled vascular imaging dataset in PAH, demonstrated improved pulmonary arteries + veins and parenchymal tissue(7)

Blockbuster Potential Across Multiple Pulmonary Indications with Unmet Need

- PAH: underserved, incurable rare disease where standard of care is to stack vasodilators(8,9)
- Differentiated anti-fibrotic evidence from Phase 2 + 3 support potential for related indications like PH-ILD and IPF(7,10,11)
- PH-ILD: only one approved mechanism, ~2x PAH population, among deadliest orphan lung diseases (~23% 5-year survival)(12,13,14)

(1): 390 WHO FC II-III patients on background therapy; 55% on triple/quadruple, 61% on a prostacyclin. Double-blind, placebo-controlled; 197 seralutinib / 193 placebo; up to 48 weeks on background PAH therapy. (2): Week 24 median +28.2 m seralutinib vs. +13.5 m placebo (placebo-adjusted +13.3 m; p=0.0320); an above-expectation placebo response compressed the delta versus the prespecified alpha of 0.025. Hodges-Lehmann estimate +13.3 m; Week 48 6MWD continued to improve on seralutinib. (3): Prespecified intermediate/high-risk subgroup (n=234) placebo-adjusted 6MWD +20.0 m (p=0.0207); all four key secondaries favored seralutinib; directionally aligned with Phase 2 TORREY; key secondaries: NT-proBNP, REVEAL Lite 2 reduction, clinical improvement, TTCW. Subgroup: baseline REVEAL Lite 2 ≥6. Nominal p-values, unadjusted for multiplicity. (4): Safety consistent with prior experience; TEAEs 86.5% vs. 80.5%; SAEs 16.0% vs. 18.9%; cough 37.0%; transaminase ≥3× ULN 13% vs. 1%; NDA submission targeted September 2026 under FDA's one-adequate-and-well-controlled-study-plus-confirmatory-evidence pathway; potential approval 3Q 2027. (5): Galkin A et al. Eur Respir J. 2022;60:2102356. (6): Sitbon O et al. Adv Ther. 2025;42:4208-4227. (7): Gossamer Bio. PROSERA Phase 3 CT-FRI Substudy. 2026. Data on file. (8): Humbert M et al. Eur Heart J. 2022;43:3618-3731. (9): Chin KM et al. Eur Respir J. 2024;64:2401325. (10): Ghofrani HA et al. Am J Respir Crit Care Med. 2024;209:A7383. (11): Sitapara R et al. CHEST Annual Meeting poster. (12): FDA, Tyvaso and Yutrepia prescribing information. (13): Based on internal company estimates; PH-ILD prevalence is estimated to be approximately 2× that of PAH. (14): Gall H et al. J Heart Lung Transplant. 2017;36:957-967. WHO FC = World Health Organization functional class; REVEAL Lite 2 = Registry to Evaluate Early and Long-term PAH Disease Management Lite 2 risk score; 6MWD = 6-minute walk distance; NT-proBNP = N-terminal pro-B-type natriuretic peptide; TTCW = time to clinical worsening; TEAE = treatment-emergent adverse event; SAE = serious adverse event; ULN = upper limit of normal; NDA = new drug application; TKI = tyrosine kinase inhibitor; OLE = open-label extension; PVR = pulmonary vascular resistance; MoA = mechanism of action; CT = computed tomography; FRI = functional respiratory imaging; PH-ILD = pulmonary hypertension associated with interstitial lung disease; IPF = idiopathic pulmonary fibrosis.

Recent Updates Since Phase 3 Topline Strengthened Gossamer Story

Repurchased Seralutinib WW Rights
Recaptures economic opportunity and increases strategic optionality

Type B Pre-NDA Meeting with FDA
FDA meeting minutes provide greater clarity on the NDA review path, with statistical significance and treatment effect magnitude to be evaluated during review

Strengthened Capital Structure
Significantly reduced debt obligations through convertible note exchange with equitization without giving up existing capital and secured additional capital

Revised FDA Guidance
Revised FDA guidance places greater emphasis on the totality of evidence and regulatory flexibility in serious diseases

Positive PROSERA Phase 3 FRI Substudy Topline Results
Evidence of reverse remodeling across the arterial, venous and parenchymal space – first of its kind in PAH

Clinical Endpoint Results from Randomized Controlled Clinical Trials in PAH

- Data across trials broadly suggests the treatment effect of seralutinib in PAH
- Under the new FDA guidance, proposing "substantial evidence of effectiveness" plan to include evidence from:
  - Phase 3 PROSERA subgroup analyses
  - Phase 2 TORREY
  - preclinical evidence
  - translational medicine, including biomarker data

Endpoint	PROSERA Overall	PROSERA Subgroup *Prespecified · Higher-Risk* REVEAL Lite 2 ≥6	TORREY Overall	TORREY Subgroup *Prespecified · Higher-Risk* REVEAL 2 ≥6	Phase 1b
N	390	234	86	37	8
Double Blind Duration	24 - 48 wk	24 - 48 wk	24 wk	24 wk	2 wk
PAH Background Therapy	PDE5 + ERA + Prostacyclin + Sotatercept	PDE5 + ERA + Prostacyclin + Sotatercept	PDE5 + ERA + Prostacyclin	PDE5 + ERA + Prostacyclin	PDE5 + ERA + Prostacyclin
6MWD (Wk 24) Measure of functional exercise capacity via 6MWT	◆ Primary +13.3 m p = 0.0320	◆ Primary +20.0 m p = 0.0207	○ Key Secondary +6.5 m p = 0.5972	○ Key Secondary +21.9 m p = 0.2482	—
PVR (Wk 24) Measure of pulmonary vascular resistance via RHC	—	—	◆ Primary 14.3% reduction p = 0.031	◆ Primary 23% reduction p = 0.0134	—
NT-proBNP (Wk 24) Blood marker for heart failure	○ Key Secondary −120.4 ng/L p = 0.0002	○ Key Secondary −265.8 ng/L p = 0.0002	−408.3 ng/L p = 0.0012	−732 ng/L p = 0.0002	—
Risk score (Wk 24) Composite risk calculator predicting survival using multiple clinical variables	○ Key Secondary OR 1.42 p = 0.0877	○ Key Secondary OR 2.03 p = 0.0083	—	—	—
Clinical Improvement Based on clinician-assessed grading of symptom severity (I–IV) based on physical activity limitations	○ Key Secondary OR 1.64 p = 0.0812	○ Key Secondary OR 3.32 p = 0.0101	—	—	—
TTCW Time to first clinical worsening event	○ Key Secondary HR 0.829 p = 0.5621	○ Key Secondary HR 0.744 p = 0.4360	—	—	—
Echo ultrasound imaging of heart structure and function	—	—	6 parameters (incl. RAA, RVFWS, PA Com.) showed improvement (p < 0.05) v. pbo	—	—
Substudy: CT FRI CT-based computational imaging of lung blood flow and vascular/airway remodeling	Arteries, veins & parenchyma saw improvement (p < 0.05) vs pbo (n = 162)	—	Arterial pruning p = 0.028 (n = 19)	—	—
Open Label Extension (OLE)	Ongoing	—	Patients on drug for 72 weeks showed a doubling in PVR reduction at week 72 vs week 24 and continued improvement in 6MWD	—	1 of 2 OLE patients remains on therapy (5+ years)

All endpoints are prespecified. Every depicted endpoint is directionally favorable. ◆ primary endpoint · ○ key secondary endpoint. — = not applicable. All PROSERA p-values are nominal, aside from the primary endpoint, 6MWD, at week 24 in the overall patient population. All TORREY p-values below 0.05 are considered statistically significant. HR = hazard ratio; OR = odds ratio; FC = functional class; WHO = World Health Organization; TTCW = time to first clinical worsening; PDE5 = phosphodiesterase type 5 inhibitor; ERA = endothelin receptor antagonist; RHC = right heart catheterization; REVEAL Lite 2 = Registry to Evaluate Early and Long-term PAH Disease Management Lite 2 risk score; NT-proBNP = N-terminal pro-B-type natriuretic peptide; OLE = open-label extension; pbo = placebo; PVR = pulmonary vascular resistance; 6MWD = six-minute walk distance; CT FRI = computed tomography functional respiratory imaging; RAA = right atrium area; RV FWS = right ventricle free wall strain; PA Com. = pulmonary arterial compliance.

A Defined Regulatory Path: Substantial Evidence of Effectiveness

Regulatory Framework	• Pursuing an NDA under the FDA framework of one adequate and well-controlled clinical investigation (PROSERA) plus confirmatory evidence (TORREY) • Gossamer proposal: PROSERA provides the primary evidence of efficacy on functional capacity; TORREY provides objective confirmatory evidence across hemodynamics and right-heart function • Gossamer proposal: Additional clinical, biomarker, imaging, mechanistic and long-term data support the consistency, biological plausibility and durability of the observed treatment effect • Supported by the seriousness of PAH, persistent unmet need and seralutinib's distinct mechanism • Currently targeting a broad PAH population; approval, as for all therapies, rests on a favorable benefit-risk; we believe the consistency and magnitude of benefit in higher-risk patients underpins clinical meaningfulness
FDA Engagement	• In-person Type B Pre-NDA meeting held in mid-June 2026 • Based on FDA feedback, we plan to submit NDA with our existing clinical package • Discussion addressed the sufficiency of efficacy and safety package for submission and substantive review • FDA provided feedback on the key elements of the planned submission, while final approvability will be determined through review of the complete NDA
Anticipated Timeline	• Targeting NDA submission in September 2026 • Potential FDA decision as early as the third quarter of 2027, if the process stays on track

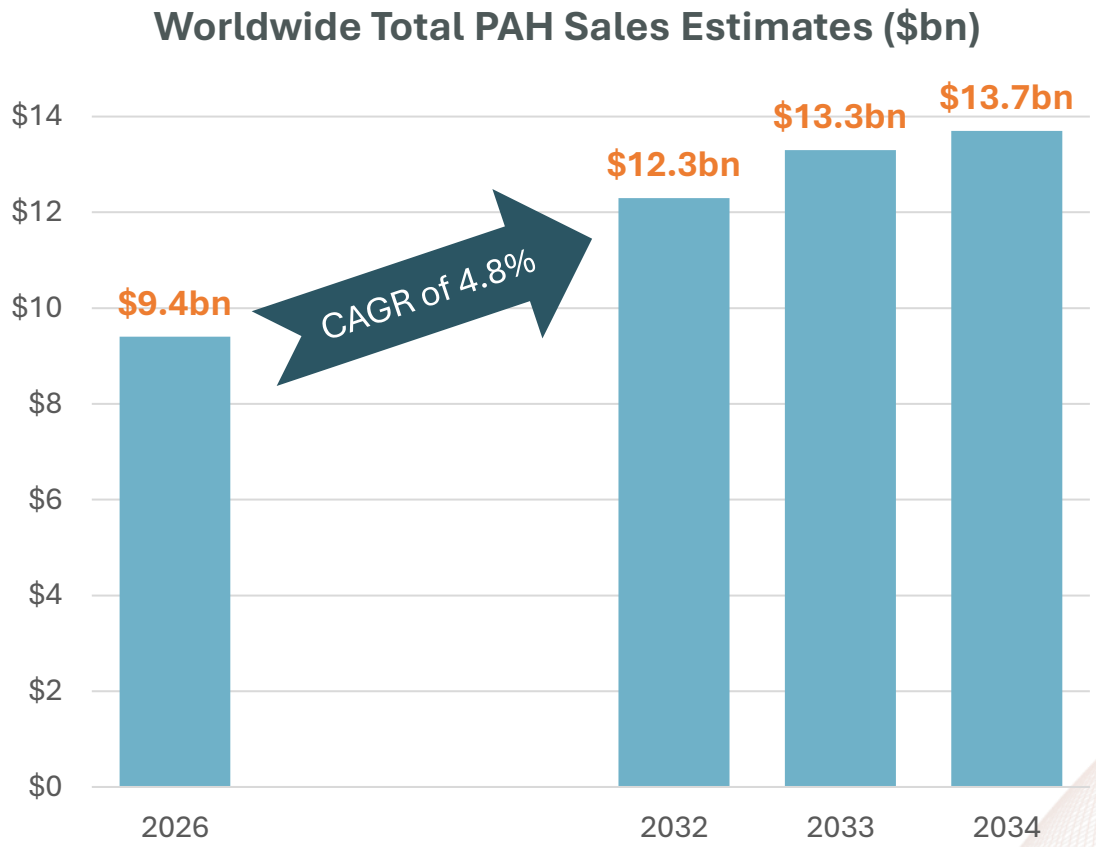
Gossamer Bets on Seralutinib: Reacquires Worldwide Rights and Substantially Recaptures Global Economics

- Reflects Gossamer's conviction in seralutinib and its decision to invest behind the program as it approaches a planned NDA submission in PAH
- Reacquired worldwide development and commercial rights to seralutinib, including full control across PAH, PH-ILD and potential future indications
  - Replaces the prior 50/50 U.S. profit share and ex-U.S. royalty structure with defined, success-based obligations
  - Gossamer retains the substantial majority of worldwide economics, while Chiesi remains entitled to a capped royalty and specified success-based milestone payments
  - No upfront cash payment by Gossamer; Chiesi will make a $5 million payment to Gossamer at signing
  - Capped royalty terminates once the agreed cap is reached, providing Gossamer with increasing participation in the program's long-term value
- Consolidates global decision-making under Gossamer across development, regulatory, manufacturing, commercialization, pricing and lifecycle strategy

Positions Gossamer shareholders to participate more fully in seralutinib's potential global upside

Blockbuster Potential in a Market Built for Disruption

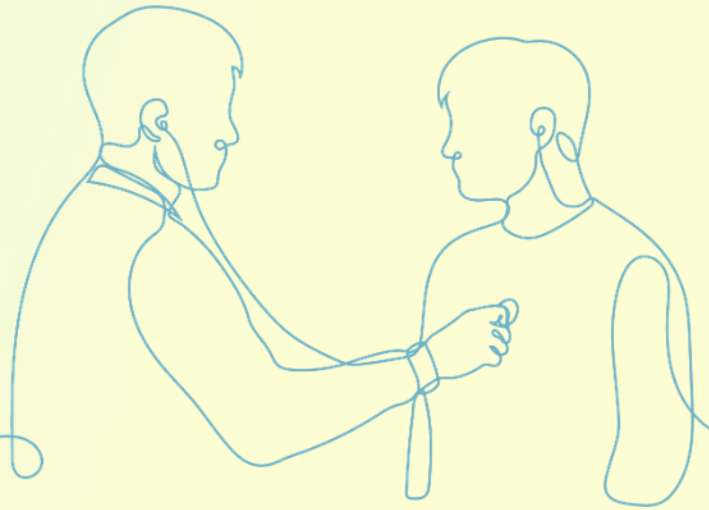
- Consensus PAH estimates support continued market growth through 2034
- High unmet need persists despite a crowded treatment landscape within most existing classes of therapy[1,2]
- Most approved therapies effect vasodilation and are not directed at reversing underlying disease biology[1,2]
- Vasodilators are increasingly going generic, while prostanoid innovation is heavily concentrated around delivery and formulation, often with existing molecules[3,4]
- Treatment burden supports demand for tolerable, add-on innovation[4,5]



As a purpose built, potential first-in-class therapy with a differentiated mechanism that is designed to address the underlying biology, we believe that seralutinib has peak sales potential of $2bn+ in PAH WW

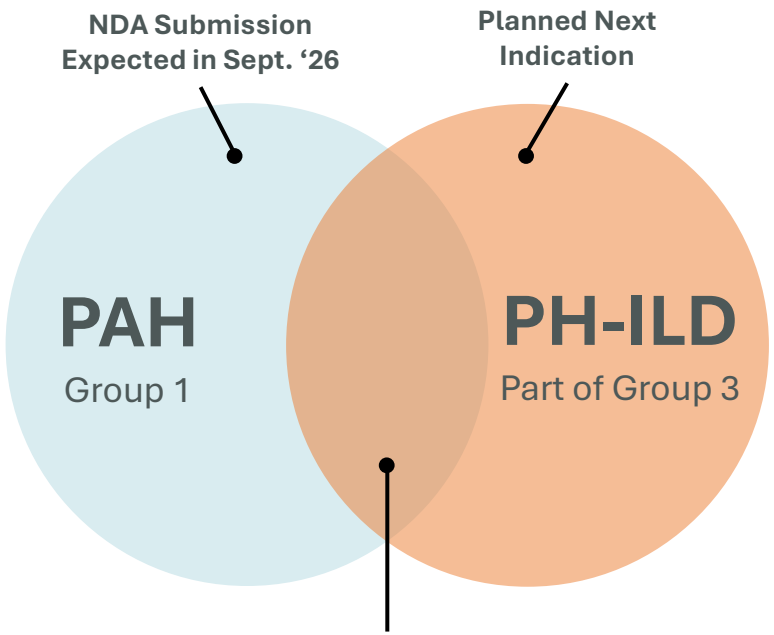
Consensus TAM estimates consolidated from research reports. (1): Chin KM et al. Eur Respir J. 2024;64:2401325. (2): Shah AJ et al. Int J Mol Sci. 2023;24:5850. (3): FDA Orange Book; FDA-approved PAH product prescribing information. (4): Lang IM, Gaine SP. Eur Respir Rev. 2015;24:630-641. (5): Elwing JM et al. Pulm Circ. 2025;15:e70098. TAM = total addressable market; WW = worldwide; CAGR = compound annual growth rate. Potential for >$2 billion in peak annual sales supported by a large addressable patient population, precedent peak sales in PAH, and proprietary epidemiology-based market assessments.

II. PAH, Mechanism & Differentiation



Pulmonary Hypertension: Encompasses a Range of Serious Conditions Driven by Elevated Pulmonary Pressure

Seralutinib is initially targeting Group 1 PH and a subgroup of Group 3 PH



Shared Features of PAH + PH-ILD[1,2]

- Elevated pulmonary pressures
- Right ventricular dysfunction
- Leads to progressive decline and elevated mortality if unmanaged

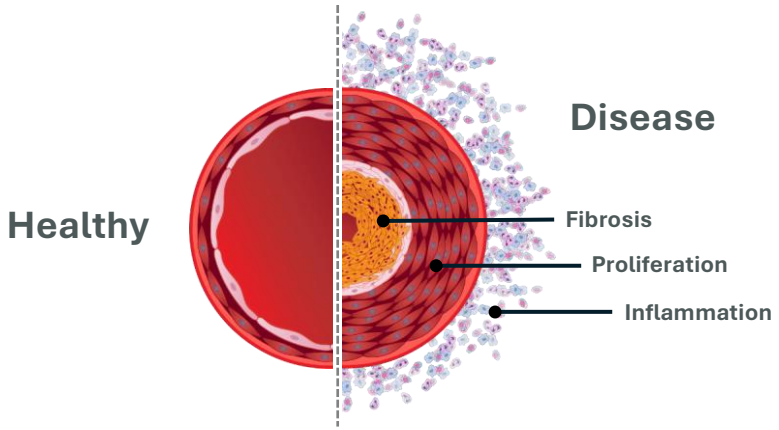
WHO groups PH into 5 Distinct Categories

WHO GROUP	CAUSE OF PH	DESCRIPTION
Group 1	PH due to Pulmonary Arterial Hypertension (PAH)	Arteries in the lungs become narrowed, thickened, or stiff (includes idiopathic, heritable, and due to drugs/toxins)
Group 2	PH due to Left Heart Disease	Problems with how the heart squeezes or relaxes, or problems with the valves on the left side of the heart
Group 3	PH due to lung disease or hypoxia, incl. PH-ILD	Lung arteries can tighten so much that blood only flows to the best-ventilated areas, causing high pressure throughout the lungs
Group 4	PH due to Chronic Blood Clots in Lungs	This can lead to scar tissue in the blood vessels of the lungs, which blocks normal blood flow and makes the right side of the heart work harder
Group 5	PH due to Unknown Causes	PH is secondary to other rare conditions and blood disorders

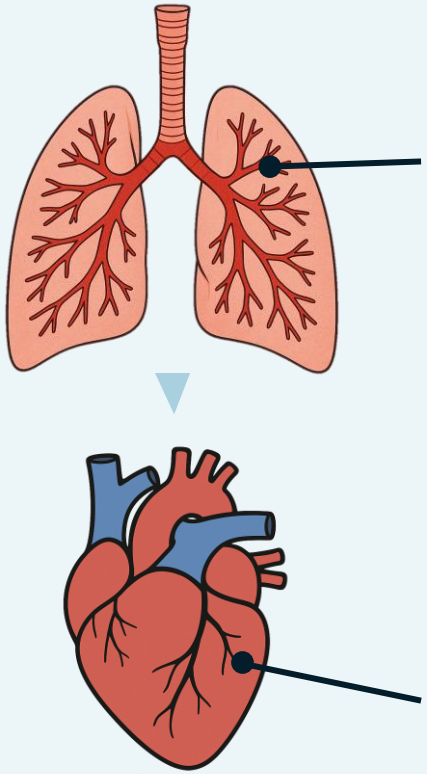
(1): Humbert M et al. 2022 ESC/ERS Guidelines. Eur Heart J. 2022;43:3618-3731. (2): Gall H et al. J Heart Lung Transplant. 2017;36:957-967. PH = pulmonary hypertension; PAH = pulmonary arterial hypertension; PH-ILD = pulmonary hypertension associated with interstitial lung disease; WHO = World Health Organization; ESC = European Society of Cardiology; ERS = European Respiratory Society.

Hypertension in the Pulmonary Vasculature of the Lungs Strains the Heart, Leading to Heart Failure & Death

- PAH is a **rare, progressive, incurable, and often fatal cardiopulmonary disease** affecting approximately 50,000 patients in the US[(1)]
- Disease is **driven by pathological vascular remodeling**, where small arteries in the lungs become narrowed, thickened, and stiff, increasing PVR, **straining the right side of the heart, and potentially leading to right heart failure and death**



Effects of PAH



Inflammation and Thrombosis
Elevated inflammation and thrombosis promote vascular remodeling and disease progression

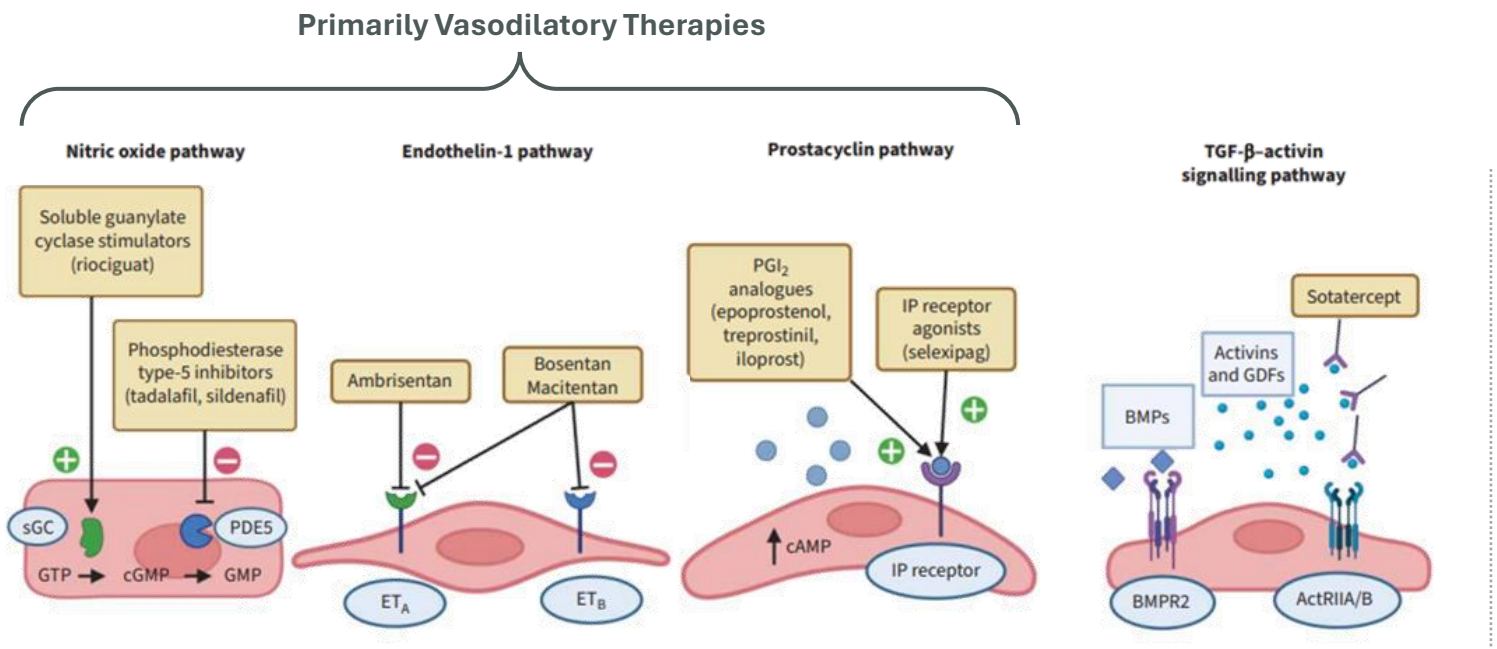
Fibrosis and Vascular Stiffening
Excessive cell growth, inflammation, and collagen buildup cause vessel wall stiffening and fibrosis, driving disease progression

Smooth Muscle Cell Hyperproliferation
Abnormal cell growth, electrical changes, and metabolic dysfunction drive vessel narrowing and increased pulmonary pressure

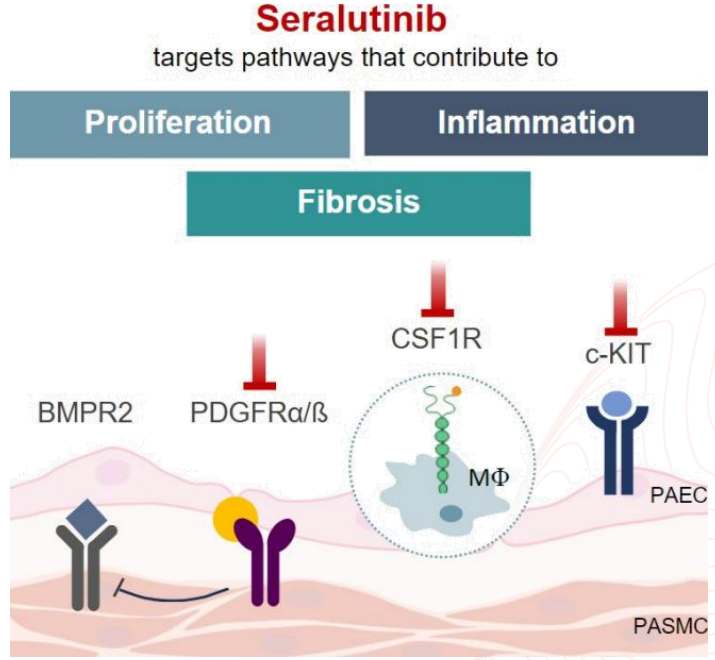
RV-RA Uncoupling and RV Failure (Leads to Death)
In PAH, metabolic dysfunction, inflammation, and oxidative stress impair right heart function, while cell loss and reduced contractility drive RV failure and poor outcomes in PAH

(1): Institute for Clinical and Economic Review (ICER). Report at a Glance: Pulmonary Arterial Hypertension (PAH). January 2024. Humbert M, et al. Circulation. 2014;130(24):2189–2208. Maron BA, et al. Am J Respir Crit Care Med. 2021;203(12):1472–1487. RA = right atrium; RV = right ventricle; PVR = pulmonary vascular resistance.

Seralutinib is A Potential First-In-Class TKI for PAH



Approved Mechanisms



Phase 3 Complete

Targeting PDGFRα/β, CSF1R and c-KIT, seralutinib has demonstrated, in pre-clinical models of PAH, a reversal of pulmonary vascular remodeling, improved hemodynamic parameters, increased lung BMPR2, and reduced circulating NT-proBNP

Source: Galkin A, Sitapara R, Clemons B, et al. Inhaled Seralutinib Exhibits Potent Efficacy in Models of Pulmonary Arterial Hypertension. European Respiratory Journal. 2022;60(6):2102356. doi:https://doi.org/10.1183/13993003.02356-2021.

TKI = tyrosine kinase inhibitor; sGC = soluble guanylate cyclase; GTP = guanosine triphosphate; cGMP = cyclic guanosine monophosphate; PDE5 = phosphodiesterase type 5; PGI_2 = prostacyclin (prostaglandin I_2); cAMP = cyclic adenosine monophosphate; IP receptor = prostacyclin receptor; ET A = endothelin receptor type A; ET B = endothelin receptor type B; BMP = bone morphogenetic protein; BMPR2 = bone morphogenetic protein receptor type 2; GDF = growth differentiation factor; ActRIIA/B = activin receptor type IIA and type IIB; PDGFR = platelet-derived growth factor receptor; CSF1R = colony-stimulating factor 1 receptor; c-KIT = mast/stem cell growth factor receptor (KIT); MΦ = macrophage; PAEC = pulmonary artery endothelial cell; PASMC = pulmonary artery smooth muscle cell; NT-proBNP = N-terminal pro-brain natriuretic peptide.

Mix-and-Match: Current PAH Treatment Paradigm

	Tyrosine Kinase Inhibitor	Activin Ligand Trap	Established Vasodilatory Therapies		
			PDE5i / sGC	ERA	Prostanoid
			Standard of Care Dual Frontline Therapy[7]		
Brand (Generic)	Seralutinib	Winrevair (sotatercept)	Revatio (Viagra, sildenafil); Adcirca (Cialis, tadalafil); Adempas (riociguat)	Opsumit (macitentan); Letairis (ambrisentan); Tracleer (bosentan)	Tyvaso, Yutrepia, Remodulin, Orenitram (treprostinil); Flolan (epoprostenol); Uptravi (selexipag); Ventavis (iloprost)
First Class Approval	Investigational[1]; Ph. 3 Complete	2024[4]	1998 (ED, PDE5i) 2005 (PAH, PDE5i)[8]	2001[9]	1995[10]
Route	Inhaled[1]	SubQ[4]	Oral[8]	Oral[9]	Inhaled, IV, SubQ, Oral[10]
Class of Mechanism	TKI inhibiting PDGFRα/β + CSF1R + c-KIT[1]	Ligand trap (BMP/activin rebalance)	Vasodilator	Vasodilator	Vasodilator
Generics Available in Class	No	No	Yes	Yes	Yes
Site of Action	Lung-targeted[1]	Systemic	Systemic	Systemic	Systemic (IV, SubQ, Oral); Lung-targeted (inhaled)
Notes	• Only Phase 3 PAH therapy with reported results in patients receiving up to four background therapies[2] • Liver-enzyme elevations were considered immune-mediated and resolved without sequelae after discontinuation[3]	• Bleeding, telangiectasias, increased hemoglobin • Pericardial effusions prompted early termination of cibotercept development in PAH[5] • STELLAR CTD subgroup showed an attenuated 6MWD effect[6]	• Backbone; generally well-tolerated; headache, flushing, dyspepsia, hypotension[8]	• Bosentan retains REMS controls related to hepatotoxicity; edema; Embryo-fetal toxicity warnings remain class wide[9]	• Headache, flushing, jaw pain, GI distress, site pain, and infection line risk[10] • Tyvaso, Yutrepia are only FDA-approved PH-ILD therapies[10]

PAH treatment is additive across pathways; initial combination therapy with risk-guided escalation, up to four therapies, is standard[7]; While patients may switch agents within a class, an entire therapy class is rarely removed absent a safety or tolerability issue

(1): Chin KM et al. Eur Respir J. 2024;64:2401325; Humbert M et al. Eur Respir J. 2023;61:2200879. (2): FDA, Revatio, Adcirca and Adempas prescribing information; FDA Orange Book. (3): FDA, Tracleer, Opsumit and Letairis prescribing information; FDA, "ERA REMS Information," 2025. (4): FDA, Flolan, Remodulin, Tyvaso, Yutrepia, Uptravi and Ventavis prescribing information. (5): FDA, Winrevair prescribing information, rev. 2025; Hoeper MM et al. N Engl J Med. 2023;388:1478-90. (6): Hoeper MM et al. N Engl J Med. 2023;388:1478-90, suppl. fig. S4. (7): Palazzini M et al. J Scleroderma Relat Disord. 2018;3:39-42. (8): Galkin A et al. Eur Respir J. 2022;60:2102356; Frantz RP et al. Lancet Respir Med. 2024;12:523-34; NCT05934526. (9): Ghofrani HA et al. "Phase 3 PROSERA Trial." ATS 2026; Gossamer Bio, PROSERA topline results, Feb. 23, 2026. (10): Gossamer Bio, PROSERA safety analysis; company-reported, first public disclosure, July 2026. (11): Keros Therapeutics, "Additional Update on the Phase 2 TROPOS Trial," Jan. 15, 2025; "TROPOS Topline Data," May 29, 2025. PDE5i = phosphodiesterase type 5 inhibitor; sGC = soluble guanylate cyclase; ERA = endothelin receptor antagonist; TKI = tyrosine kinase inhibitor; REMS = Risk Evaluation and Mitigation Strategy; ED = erectile dysfunction; IV = intravenous; SubQ = subcutaneous; GI = gastrointestinal; BMP = bone morphogenetic protein; PDGFRα/β = platelet-derived growth factor receptor alpha/beta; CSF1R = colony stimulating factor 1 receptor; c-KIT = mast/stem cell growth factor receptor (KIT); 6MWD = 6-minute walk distance; CTD = connective tissue disease. The clinical data presented in this presentation comparing seralutinib and other products and product candidates are based on cross-study comparisons and are not based on any head-to-head clinical trials. Differences exist between trial designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable.

A Differentiated Approach for the Persistent Unmet Need in PAH

Significant Residual Disease Burden

- PAH remains progressive and incurable despite an expanding treatment arsenal
- Many patients require multiple therapies yet remain functionally limited and at risk of clinical worsening
- A need remains for therapies with meaningful activity beyond established pathways

Complex, Burdensome Treatment

- Current treatment often requires stacking multiple therapies across available pathways
- Prostacyclin therapies can require complex administration, frequent dosing or continuous infusion
- Treatment burden can complicate escalation and long-term disease management

Seralutinib: Purpose-Built for PAH

- Distinct, first-in-class mechanism targeting PDGFR, CSF1R and c-KIT
- Designed to directly address vascular remodeling, proliferation, inflammation and fibrosis
- Lung-targeted delivery through a discreet, pocket-sized inhaler
- Designed as a practical add-on to contemporary background therapy

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Sources: Chin KM et al. Eur Respir J. 2024;64:2401325; Rahaghi FF et al. Lancet Respir Med. 2025;13:364-370; Galkin A et al. Eur Respir J. 2022;60:2102356; Lang IM, Gaine SP. Eur Respir Rev. 2015;24:630-641.
PAH = pulmonary arterial hypertension; PDGFR = platelet-derived growth factor receptor; CSF1R = colony stimulating factor 1 receptor; TKI = tyrosine kinase inhibitor.

III. PROSERA Phase 3 Pivotal Data

PROSERA Phase 3:
Designed to Test Seralutinib in Contemporary PAH

- Large, global Phase 3 trial evaluating seralutinib 90 mg BID versus placebo, added to existing PAH therapy
- 390 WHO FC II–III patients with substantial residual disease despite treatment
  - Highly treated population: ~55% on at least three background therapies and ~61% on a prostacyclin
- Broad efficacy signal: Improvement across exercise capacity, NT-proBNP, clinical improvement, risk score and time to clinical worsening
- Greater effects in higher-risk patients: Stronger and more consistent benefit in the prespecified REVEAL Lite 2 ≥6 population
- Safety supported add-on use: Serious adverse events were numerically lower than placebo; key risks were generally identifiable and manageable
- Conclusion: Results support meaningful activity on top of contemporary therapy, with the greatest benefit in patients with the greatest unmet need

PROSERA Demonstrated Consistent Seralutinib Activity Across the Overall Population, Higher-Risk Patients and Key Geographies

Primary Endpoint | 6-minute walk distance at week 24

Seralutinib

+28.2 meters

Median change from baseline

Placebo-Adjusted 6MWD Improvement

+13.3 meters

p = 0.0320 | prespecified α = 0.025

Hodges-Lehmann estimate; 97.5% CI: –0.6 to 27.2

Placebo

+13.5 meters

Median change from baseline

Greater Benefit In Higher-risk Patients

+20.0 meters

Placebo-adjusted 6MWD improvement

Prespecified intermediate- and high-risk subgroup
REVEAL Lite 2 ≥6; n = 234; p = 0.0207

3 of 4 key secondary endpoints achieved nominal p < 0.0125

Broad Endpoint Consistency

4 of 4 Key secondary endpoints favored seralutinib in the overall population

- ✓ Time to clinical worsening
- ✓ Clinical improvement
- ✓ NT-proBNP
- ✓ REVEAL Lite 2 risk reduction

NT-proBNP: –120.4 ng/L vs. placebo; nominal p = 0.0002

In addition to the greater effect observed in higher-risk patients, seralutinib demonstrated a +37.0 meters placebo-adjusted improvement in 6MWD at Week 24 in CTD-associated PAH (n = 87; nominal p = 0.0104), a clinically challenging population that has historically been more difficult to treat

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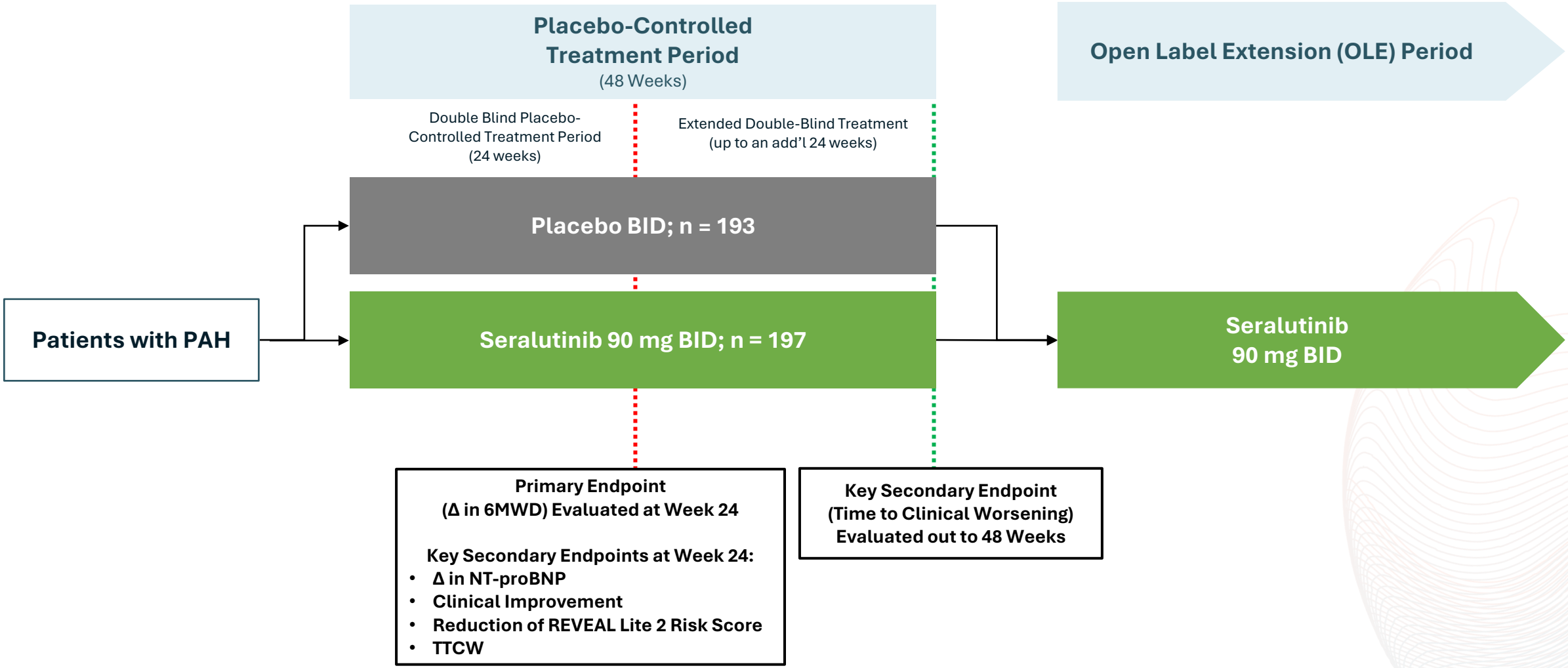
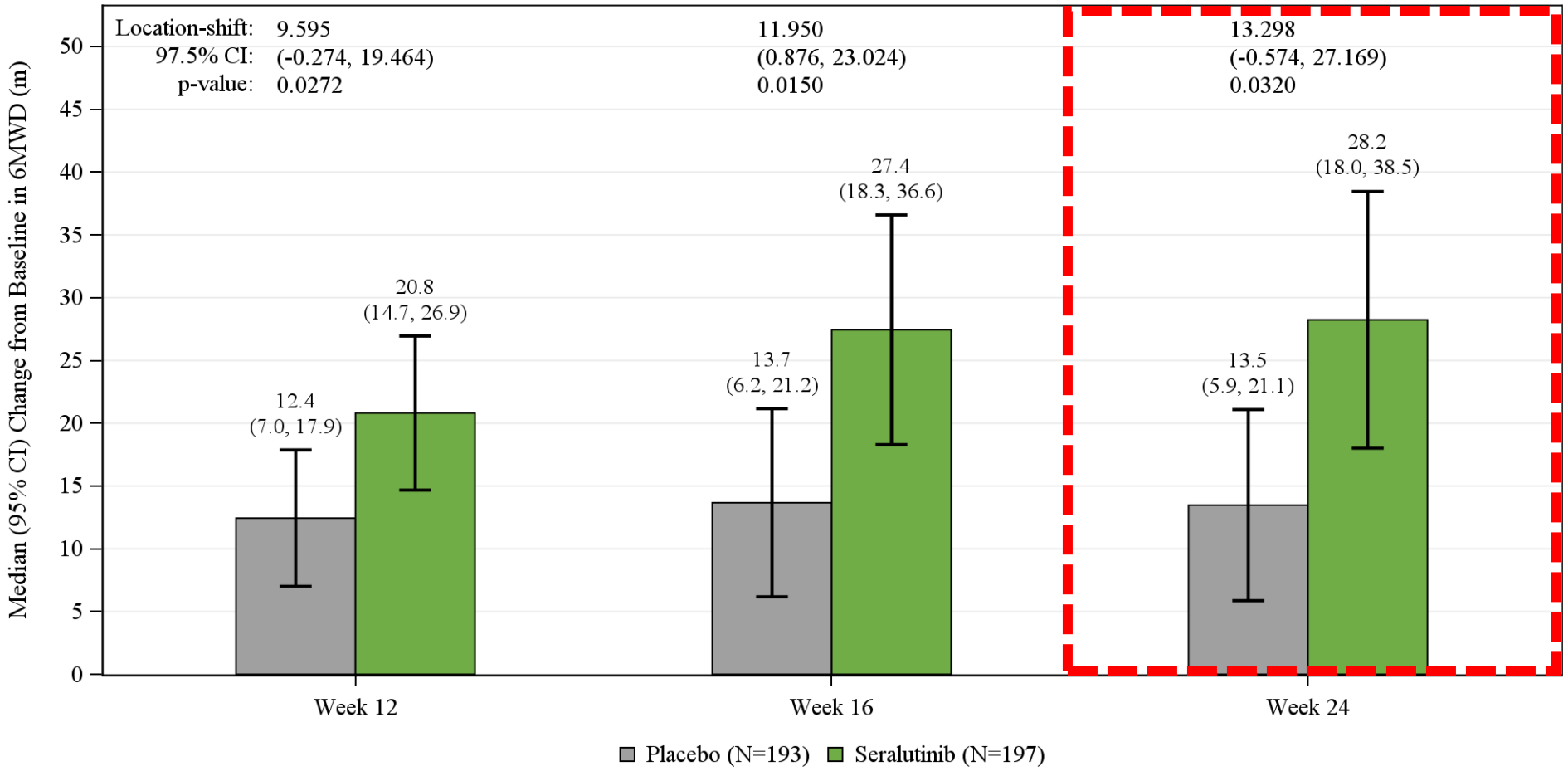
Sources: PROSERA Phase 3 topline results and supporting analyses. 6MWD = 6-minute walk distance; CI = confidence interval; NT-proBNP = N-terminal pro-B-type natriuretic peptide; REVEAL Lite 2 = Registry to Evaluate Early and Long-term PAH Disease Management Lite 2; CTD = connective tissue disease.

Phase 3 PROSERA Trial Design



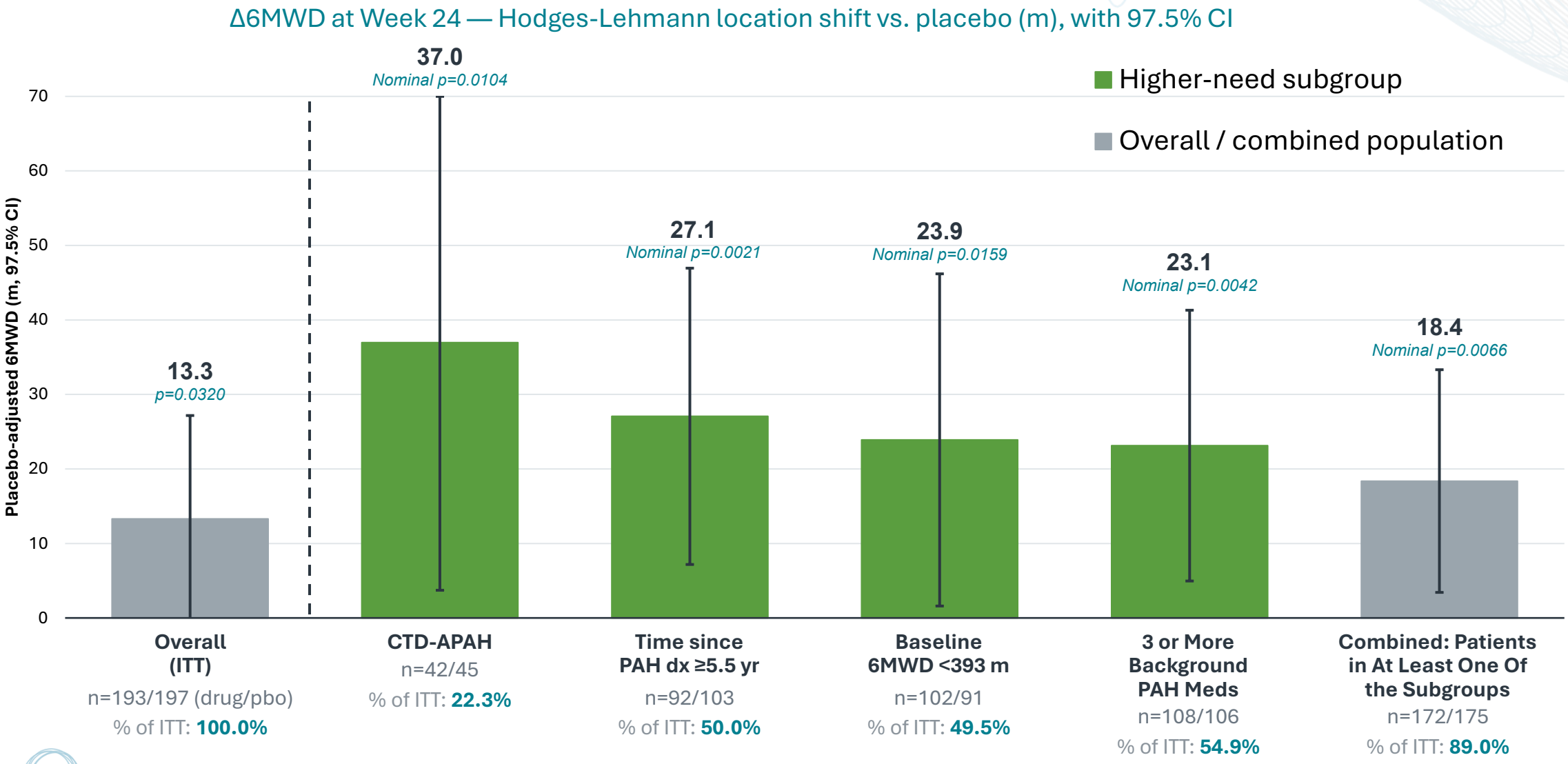
PROSERA was designed as a single pivotal trial with a prespecified stringent two-sided α = 0.025 for the primary endpoint (Δ6MWD at Week 24, ITT), using a non-parametric ARSW test with a Hodges-Lehmann treatment-effect estimate; key secondaries were tested in a fixed sequence gated on the primary.
BID = twice-daily; 6MWD = 6-minute walk distance; NT-proBNP = N-terminal pro-B-type natriuretic peptide; REVEAL Lite 2 = Registry to Evaluate Early and Long-term PAH Disease Management Lite 2 risk score;

While Separation from Placebo is Apparent, Seralutinib Did Not Meet the Prespecified α Threshold of 0.025, Largely Due to Unexpected Placebo Performance



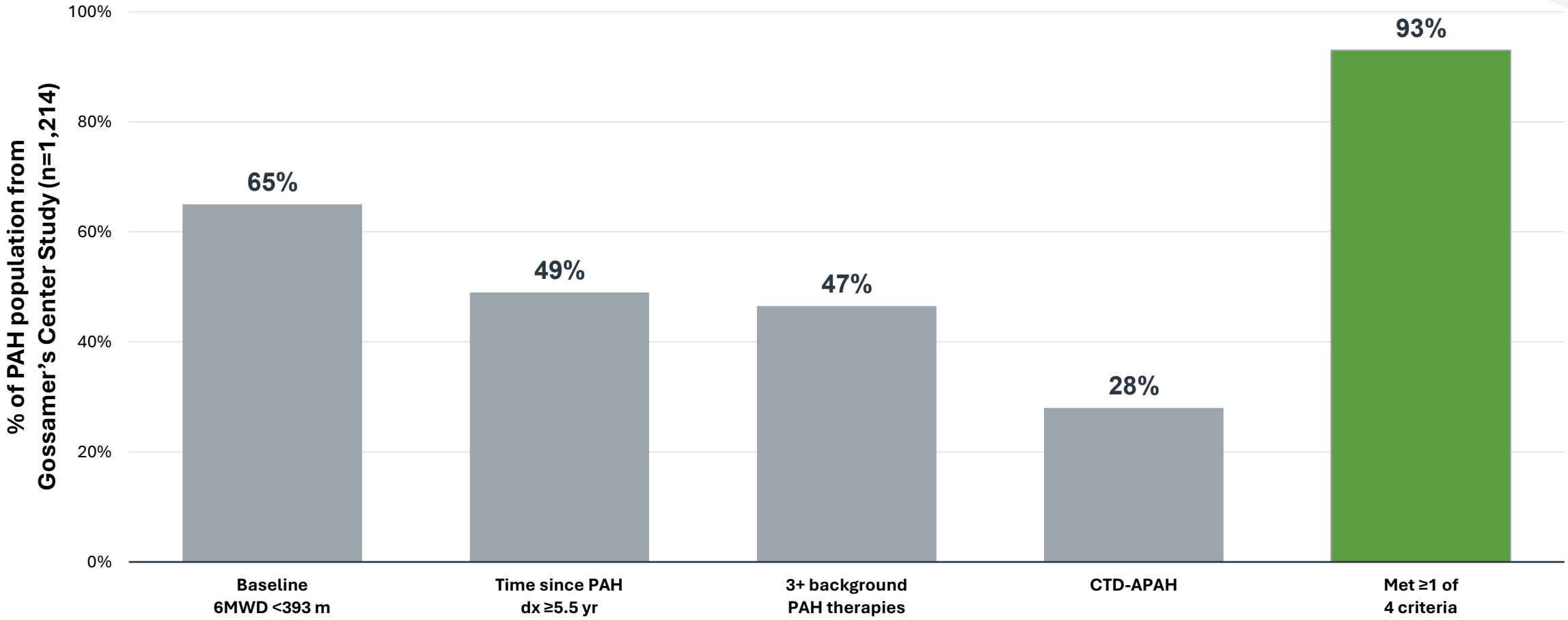
6MWD = 6-minute walk distance; CI = confidence interval.

Treatment Effect Is Amplified Across Higher-Need Prespecified Subgroups



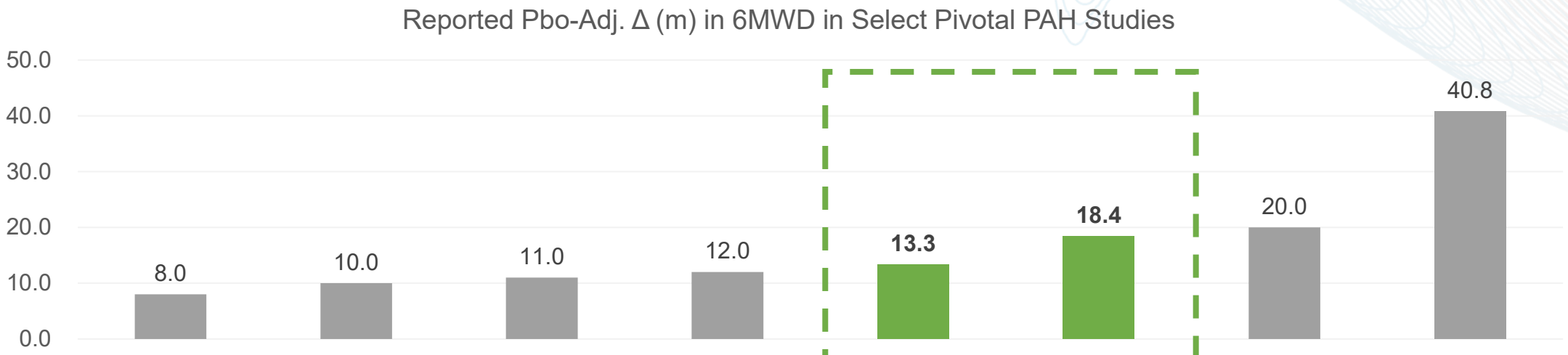
CI = confidence interval; 6MWD = 6-minute walk distance; CTD-APAH = connective tissue disease-associated PAH; ITT = intent-to-treat. % of ITT = (placebo n + seralutinib n) / 390. Combined Subgroup = 6MWD <393 m OR ≥3 PAH meds OR PAH dx ≥5.5 yr OR CTD-APAH (n=347, 89.0% of ITT; bar reflects 172/175 with MI data). Δ6MWD at Week 24, ARSW with MI.

Most PAH Patients Meet At Least One Prespecified Higher Unmet Need Criterion



Gossamer Bio. Data on file. Center Study Final Report. 2026.
Center Study: Real-world dataset of 1,214 prevalent PAH patients from seven PHA-certified centers, representing a contemporary U.S. PAH treatment population. PHA-certified centers referenced in the study population. 6MWD = 6-minute walk distance; CTD-APAH = connective tissue disease-associated PAH.

Comparison of PROSERA 6MWD Δ vs. Approved PAH Add-On Therapies



Compound	Oral Treprostinil			Selexipag	Seralutinib		Inhaled Tre	Sotatercept
Study	FREEDOM-EV[1]	FREEDOM-C2[2]	FREEDOM-C[3]	GRIPHON[4]	PROSERA (Overall)	PROSERA (Combined Higher Need Subgroups)	TRIUMPH[5]	STELLAR[6]
Timepoint	Week 24	Week 16	Week 16	Week 26	Week 24	Week 24	Week 12	Week 24
Year	2018	2010	2010	2014	2026	2026	2007	2022
Active Δ	16m	15m	14.5m	4m	28.2m	32.6m	21.6m	34.4m
Placebo Δ	8m	11m	4.8m	-9m	13.5m	13.4m	3.0m	1.0m
Background PAH Therapies	PDE5 + ERA	PDE5 + ERA	PDE5 + ERA	PDE5 + ERA	PDE5 + ERA + Prostacyclin + Sotatercept		PDE5 or ERA	PDE5 + ERA + Prostacyclin
Most Recent TTM Sales[7]		**$512mm**		**$1.9bn**	**NA**	**NA**	**$1.9bn**	**$1.7bn**

Note: Seralutinib is an investigational agent not approved for use in any jurisdiction. The clinical data presented in this presentation comparing seralutinib and other products and product candidates are based on cross-study comparisons and are not based on any head-to-head clinical trials. Differences exist between trial designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable. Pbo = placebo; Tre = Treprostinil; ERA = endothelin receptor antagonist; PDE5 = phosphodiesterase type 5 (PDE5) inhibitor. 1) White, et al. AJRCCM 2020; 2) clinicaltrials.gov/study/NCT00887978; 3) clinicaltrials.gov/study/NCT00325442; 4) Sitbon, et al. NEJM 2015; 5) McLaughlin, et al. JACC 2010; 6) Hoeper, et al. NEJM 2023; 7) SEC Filings.

Key Secondary Endpoints Favored Seralutinib in Overall Patient Population (all comparisons vs. placebo)

Change in NT-proBNP

-120.4 ng/L

(97.5% CI: -201.6, -39.1
p-value: 0.0002)

Clinical Improvement

1.6x times more likely to improve

(OR: 1.642, 97.5% CI: 0.863, 3.106
p-value: 0.0812)

Reduction in Risk Score (≥1 point)

1.4x times more likely to have reduction in risk

(OR: 1.420, 97.5% CI: 0.896, 2.248
p-value: 0.0877)

Time to Clinical Worsening

17% reduction

(HR: 0.829, 97.5% CI: 0.393, 1.708,
p-value: 0.5621)

OR = odds ratio; HR = hazard ratio; CI = confidence interval; NT-proBNP = N-terminal pro-B-type natriuretic peptide.
Note: p-values nominal.

Likewise, Enhanced Effects Were Seen in Intermediate- to High-Risk Sub-Group for Key Secondary Endpoints (all comparisons vs. placebo)

Change in NT-proBNP

-265.8 ng/L

(97.5% CI: -446.2, -85.5
p-value: 0.0002)

Clinical Improvement

3.3x times more likely to improve

(OR: 3.318, 97.5% CI: 1.167, 9.43
p-value: 0.0101)

Reduction in Risk Score (≥1 point)

2.0x times more likely to have reduction in risk

(OR: 2.033, 97.5% CI: 1.113, 3.713
p-value: 0.0083)

Time to Clinical Worsening

26% reduction

(HR: 0.744, 97.5% CI: 0.307, 1.728,
p-value: 0.4360)

OR = odds ratio; HR = hazard ratio; CI = confidence interval; NT-proBNP = N-terminal pro-B-type natriuretic peptide.
Note: p-values nominal.

IV. PROSERA Safety & Tolerability Data

Seralutinib Safety and Tolerability Data Support Potential in PAH

- Serious adverse events were numerically lower vs. placebo (16.0% vs. 18.9%) despite patients receiving seralutinib on top of up to 3 - 4 background PAH therapies (PROSERA Phase 3)
- Well-tolerated in the heavily treated PAH population (≈55% on ≥3 therapies; ≈61% on prostacyclin), supporting an add-on profile in real-world use
- No new or unexpected safety signals; adverse events were largely consistent with inhaled delivery or background standard-of-care therapies (PROSERA; consistent with prior TORREY/OLE experience)
- Manageable safety data: cough (inhaled class effect) typically mild and time-limited, and liver enzyme elevations presented with standard monitoring and reversed without sequelae after discontinuation
- Lung-targeted delivery designed to limit systemic toxicity to prevent safety issues seen with precedent TKIs in PAH (imatinib)

Overall Summary of Treatment-Emergent Adverse Events (Safety Population)

Category	Placebo (N=190) n (%)	Seralutinib (N=200) n (%)
Number of subjects with at least one:		
TEAE	153 (80.5)	173 (86.5)
Severe TEAE	30 (15.8)	30 (15.0)
TEAE leading to discontinuation of IP	11 (5.8)	30 (15.0)
AESI	12 (6.3)	41 (20.5)
SAE	36 (18.9)	32 (16.0)
Fatal TEAE	3 (1.6)	4 (2.0)
Number of SAEs	67	53

TEAE = treatment-emergent adverse event; IP = investigational product; AESI = adverse event of special interest; SAE = serious adverse event.

Incidence of SAEs by preferred term: ≥ 2 Seralutinib subjects (Safety Population)

Preferred Term	Placebo (N=190) n (%)	Seralutinib (N=200) n (%)
Number of subjects with a SAE	36 (18.9)	32 (16.0)
Pneumonia	3 (1.6)	3 (1.5)
Pulmonary arterial hypertension	5 (2.6)	3 (1.5)
Right ventricular failure	4 (2.1)	3 (1.5)
Acute kidney injury	0	2 (1.0)
Device malfunction	2 (1.1)	2 (1.0)
Lower gastrointestinal haemorrhage	0	2 (1.0)

SAE = serious adverse event.

Incidence of TEAEs by preferred term: ≥ 5% in Seralutinib arm (Safety Population)

Preferred Term	Placebo (N=190) n (%)	Seralutinib (N=200) n (%)
Number of subjects with a TEAE	153 (80.5)	173 (86.5)
Cough	26 (13.7)	74 (37.0)
Headache	27 (14.2)	32 (16.0)
Alanine aminotransferase increased	1 (0.5)	29 (14.5)
Aspartate aminotransferase increased	1 (0.5)	28 (14.0)
Nausea	20 (10.5)	24 (12.0)
Diarrhoea	26 (13.7)	23 (11.5)
Upper respiratory tract infection	10 (5.3)	17 (8.5)
Dizziness	14 (7.4)	13 (6.5)
Dyspnoea	16 (8.4)	11 (5.5)
Hypokalaemia	14 (7.4)	11 (5.5)
Nasopharyngitis	17 (8.9)	11 (5.5)
Vomiting	11 (5.8)	11 (5.5)
Influenza	8 (4.2)	10 (5.0)
Syncope	8 (4.2)	10 (5.0)

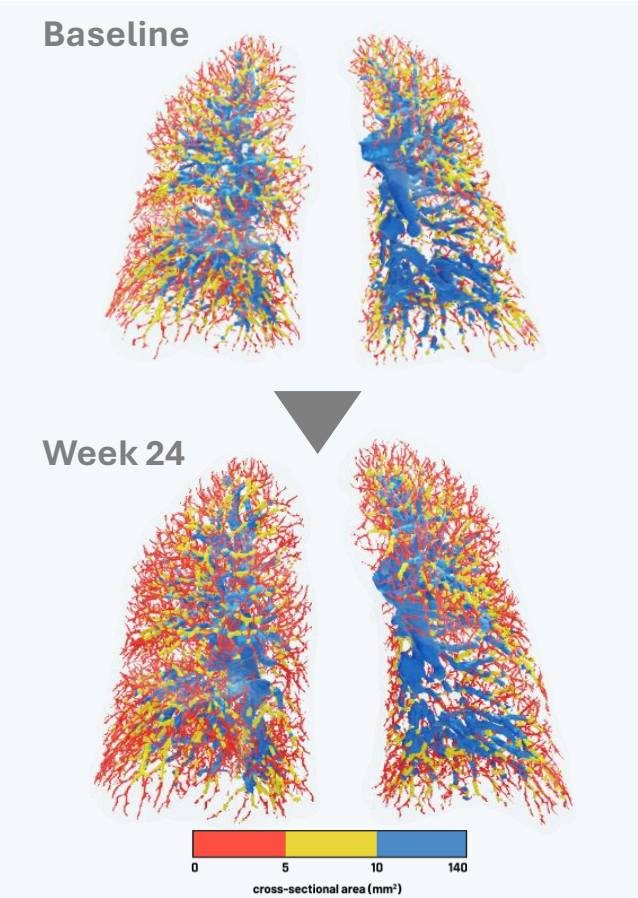
TEAE = treatment-emergent adverse event.

V. Proposed Body of Confirmatory Evidence TORREY, OLE & Preclinical

Seralutinib Demonstrated Multi-Compartment Vascular Effects

- Prespecified imaging endpoints showed statistically significant, multi-compartment effects of seralutinib across arterial, venous, and fibrosis-like parenchymal parameters
- The breadth and internal consistency of these effects exceed prior controlled PAH imaging data, including TORREY's arterial-only signal
- Imaging changes correlated more tightly with clinical endpoints (including 6MWD, NT-proBNP, REVEAL Lite 2) than in TORREY, reinforcing clinical relevance
- Pattern is biologically coherent and consistent with seralutinib's platelet-derived growth factor receptor (PDGFR) + colony stimulating factor 1 receptor (CSF1R) + c-KIT mechanism of action
- Substudy was prespecified as exploratory; all p-values are nominal and unadjusted for multiplicity

Seralutinib Patient Case Study:
Visualization of Reverse Remodeling



Color indicates vessel cross sectional area. Red denotes small-caliber vessels; blue denotes large-caliber vessels. Redistribution from blue to red is consistent with reverse remodeling. Individual result; treatment effects vary by patient.

6MWD = 6-minute walk distance; NT-proBNP = N-terminal pro–B-type natriuretic peptide; PDGFR = platelet-derived growth factor receptor; CSF1R = colony stimulating factor 1 receptor; REVEAL Lite 2 = Registry to Evaluate Early and Long-Term PAH Disease Management Lite 2 risk score; c-KIT = mast/stem cell growth factor receptor.

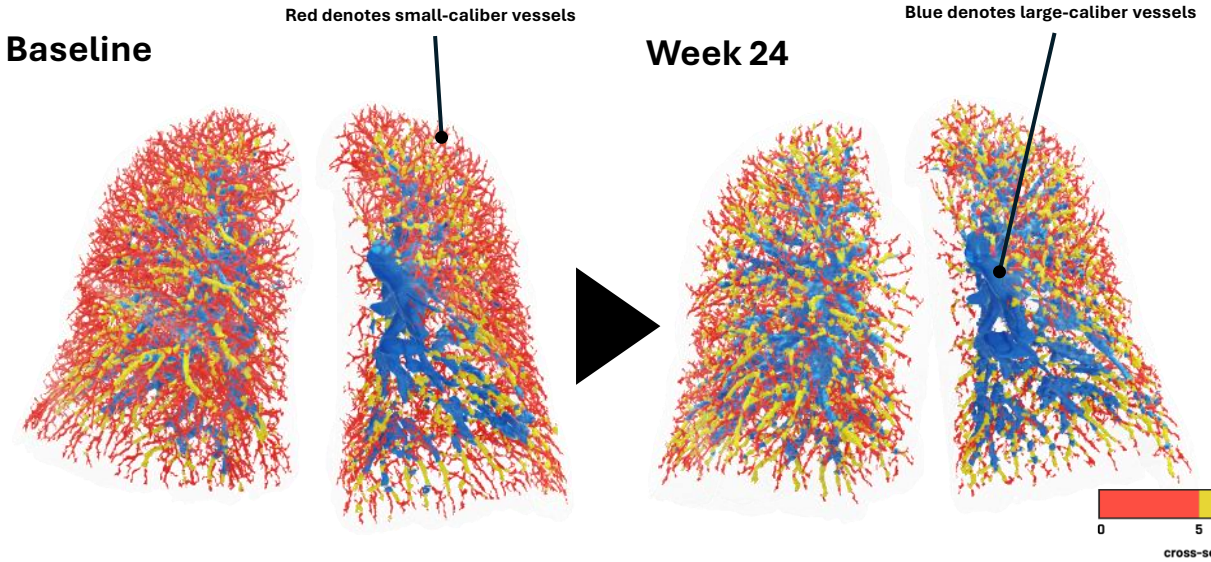
Seralutinib Demonstrated Statistically Significant Treatment Effects Across Multiple Vascular Compartments

Category	Parameter	Abbrev.	Effect	p-value	Interpretation
Arterial	Arterial vessel volume >10mm^2 / TBV	BV10A%	↓ Decrease	0.020*	Reduced proportion of large-artery volume consistent with proximal decompression
Arterial	Arterial vessel volume >10mm^2	BV10A	↓ Decrease	0.230	Reduced large-artery volume
Arterial	Small / large arterial vessel ratio	BV5A/BV10A	↑ Increase	0.556	Shift in arterial volume toward smaller vessels
Arterial	Arterial vessel volume <5mm^2	BV5A	↑ Increase	0.660	Increased small-vessel arterial volume
Parenchymal	Fibrosis-like parenchymal volume	FV	↓ Decrease	0.026*	Reduced fibrosis-like parenchymal volume
Parenchymal	Normalized fibrosis-like parenchymal volume	FVN	↓ Decrease	0.025*	Reduced proportion of fibrosis-like volume
Venous	Total venous blood volume	TVV	↑ Increase	0.016*	Increased venous filling; placebo decreased
Venous	Venous vessel volume <5mm^2	BV5V	↑ Increase	0.034*	Increased small venous vessel volume
Venous	Venous vessel volume 5–10mm^2	BV510V	↑ Increase	0.041*	Increased mid-size venous vessel volume
Venous	Venous vessel volume >10mm^2	BV10V	↑ Increase	0.188	Increased large venous vessel volume
Venous	Fractal dimension of venous vessels	FDOPV	↑ Increase	0.044*	Increased vascular branching

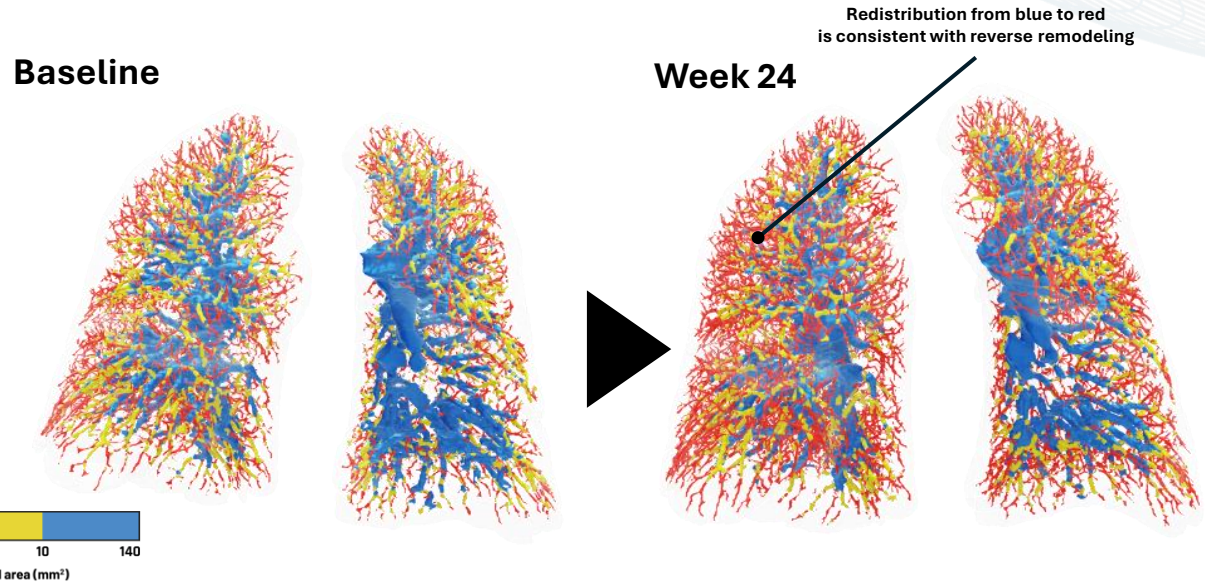
TBV = total blood volume; 6MWD = 6-minute walk distance; FDOPV = fractal dimension of pulmonary venous vessels; BV5VPR = venous vessel volume <5mm^2 / total blood volume; SAP = statistical analysis plan; FRI = functional respiratory imaging; CT = computed tomography; IPF = idiopathic pulmonary fibrosis. In addition to the 7 nominally statistically significant prespecified parameters shown above, BV5VPR (venous vessel volume <5mm^2 / TBV) also achieved nominal significance (p=0.020). Prespecified exploratory parameters per SAP v2.1; p-values are nominal and unadjusted for multiplicity. Clinical correlations shown are Spearman, segregated as baseline vs. Δ, pooled across both arms, and are descriptive rather than confirmatory. FRI fibrosis volume (FV) quantifies CT voxel-level features characteristic of fibrotic tissue, derived from a deep-learning algorithm (FibroNet) trained on confirmed IPF patient datasets. * denotes statistical significance.

Two Patients on Triple Background Therapy, Two Trajectories:
Visualization of Reverse Remodeling (Placebo v. Seralutinib)

Placebo patient



Seralutinib patient

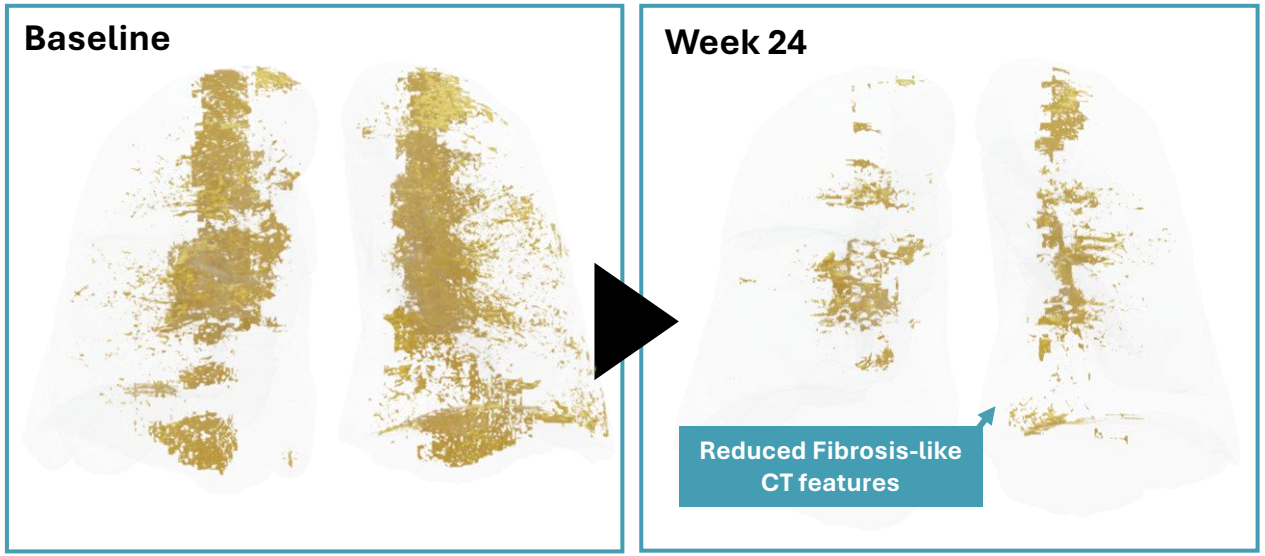


60y Female, PAH-CTD, FC 3, Triple Therapy	
Δ 6MWD	-1 m
Δ NT-proBNP	-143 ng/L
Δ BV10A, BV5A	+12.6 mL, -6.8 mL
Δ Total Venous Volume	-8.9 mL

70y Female, IPAH, FC 3, Triple Therapy	
Δ 6MWD	+55 m
Δ NT-proBNP	-324 ng/L
Δ BV10A, BV5A	-4.2 mL, +5.5 mL
Δ Total Venous Volume	+8.8 mL

IPAH = idiopathic pulmonary arterial hypertension; CTD = connective tissue disease; FC = functional class; 6MWD = 6-minute walk distance; NT-proBNP = N-terminal pro–B-type natriuretic peptide; BV10A = arterial vessel volume >10 mm^2; BV5A = arterial vessel volume <5 mm^2.
Individual patient data are illustrative and not representative of the overall study population.

Visible Reduction in Fibrosis-like Parenchymal Features Seen in PAH Patient

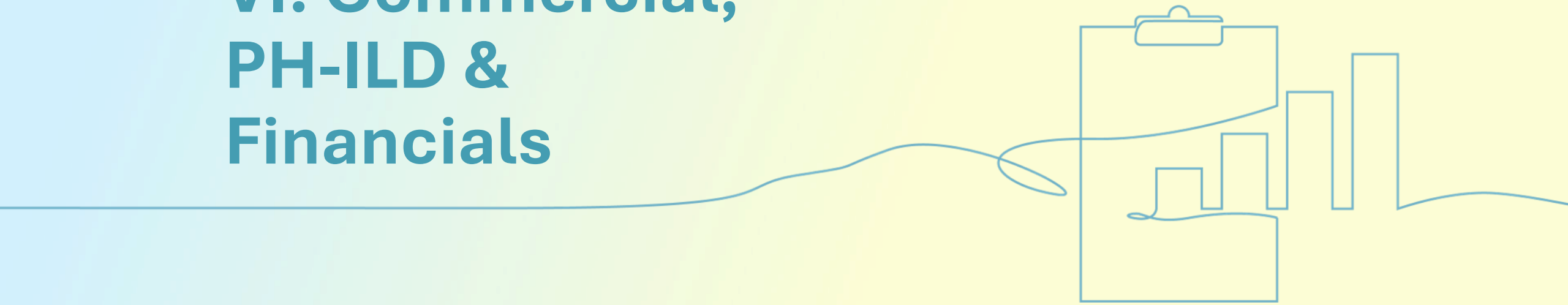


Seralutinib patient	
54yo female, IPAH, FC 3, Double Therapy	
Δ 6MWD	+73m
Δ NT-proBNP	-307 ng/L
Δ FV	-64.7 mL
Δ FVN	-1.64

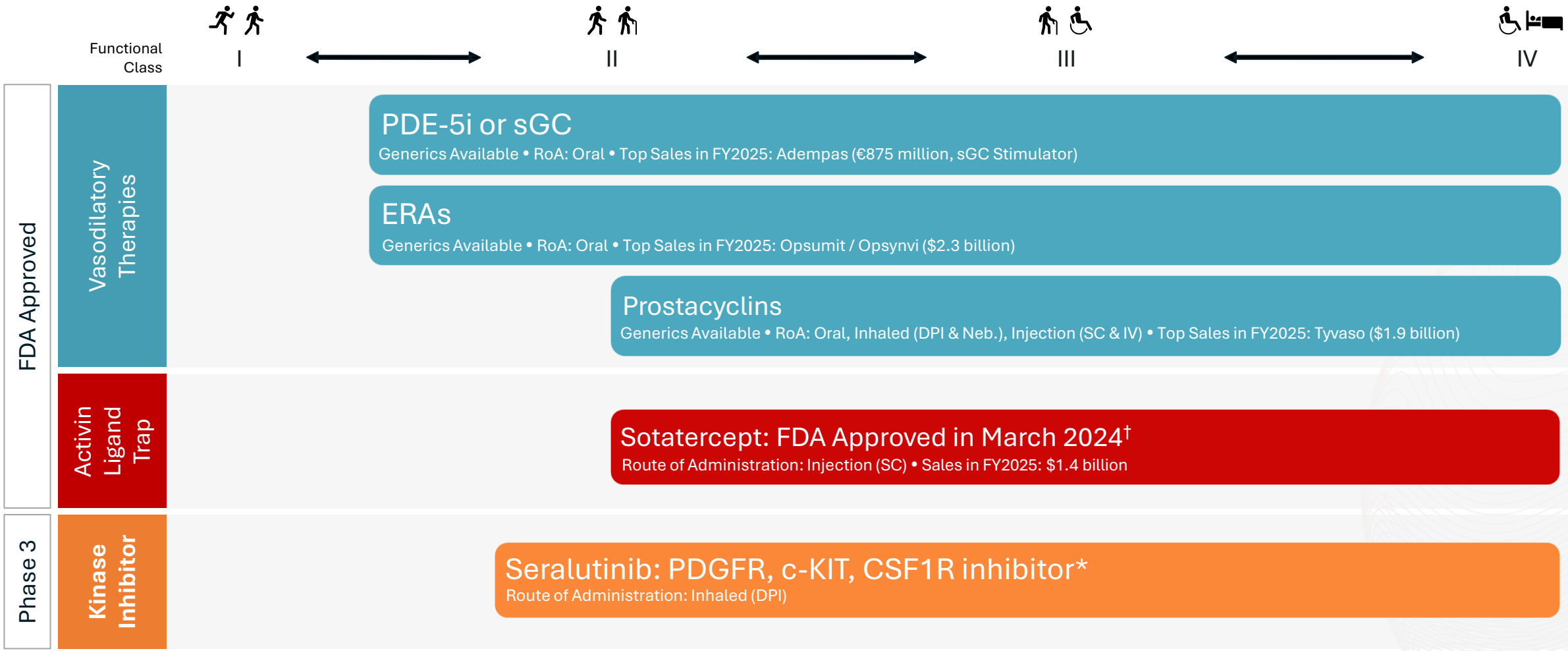
- Fibrosis volume (FV) quantified via FibroNet, a deep-learning algorithm trained on confirmed idiopathic pulmonary fibrosis (IPF) data (analogous to High Attenuation Abnormalities [HAA] in ILD imaging)
- CT detects fibrotic changes only around larger vessels; true parenchymal remodeling burden is likely underestimated, especially in the sub-CT-resolution capillary bed
- Consistent with seralutinib's PDGFR / CSF1R / c-KIT mechanism: fibroblast, macrophage and mast cell remodeling inhibition, reflected as reduced parenchymal density on CT

FV = fibrosis volume; FVN = normalized fibrosis volume; CT = computed tomography; FRI = functional respiratory imaging; PDGFR = platelet-derived growth factor receptor; CSF1R = colony stimulating factor 1 receptor; c-KIT = KIT proto-oncogene receptor tyrosine kinase; HAA = High Attenuation Abnormalities; ILD = interstitial lung disease; IPF = idiopathic pulmonary fibrosis; PH-ILD = pulmonary hypertension–interstitial lung disease; IPAH = idiopathic pulmonary arterial hypertension; FC = functional class; 6MWD = 6-minute walk distance; NT-proBNP = N-terminal pro–B-type natriuretic peptide. FRI fibrosis volume (FV) quantifies CT voxel-level high-attenuation features characteristic of fibrotic tissue. All p-values are nominal and unadjusted for multiplicity. Individual patient images are illustrative and not representative of the study population.

VI. Commercial, PH-ILD & Financials



In a Treatment Paradigm Long-Dominated by Vasodilatory Therapies, Patients Need Treatments that Address the Underlying Disease



Sources: Company financial reports.
PDE-5i = phosphodiesterase type 5 inhibitor; sGC = soluble guanylate cyclase; ERA = endothelin receptor antagonist; RoA = route of administration; DPI = dry powder inhaler; Neb. = nebulized; SC = subcutaneous injection; IV = intravenous injection; PDGFR = platelet-derived growth factor receptor; CSF1R = colony stimulating factor 1 receptor; c-KIT = mast/stem cell growth factor receptor (KIT).
†Sotatercept positioning is conjectural and subject to adjustment, given its recent entry into the market and the PAH treatment paradigm.
*Reflects potential positioning for an investigational therapy that is not yet approved and is subject to regulatory review and approval. Subject to change.

Seralutinib's Next Frontier: What is PH-ILD?

- WHO Group 3 PH refers to PH associated with lung diseases and / or hypoxia[2]
- PH-ILD is a specific subgroup within Group 3 PH, which includes PH related to IPF & PH associated with CTD-ILD[2]
- PH-ILD is characterized by pulmonary vascular changes associated with PH, in addition to thickening & scarring of the lung interstitium resulting from ILD[2]
- Tyvaso and Yutrepia are approved therapies for PH-ILD, both delivering inhaled Treprostinil; Tyvaso is administered 4 times daily; Yutrepia is administered 3 – 5 times daily [3,4]
- **PH-ILD carries poor prognosis & increased mortality rate as compared to PAH patients (40% 3-year survival rate)[5,6]**

~60-100K PH-ILD patients in US[1]

One FDA approved mechanism (treprostinil)[3,4]

Call point overlap with PAH

(1): Based on internal company estimates. Prevalence estimates of PH-ILD indicate the patient population is likely to be 1-2 times that of PAH. (2): Humbert M et al. Eur Heart J. 2022;43:3618-3731. (3): FDA, Tyvaso and Tyvaso DPI prescribing information. (4): FDA, YUTREPIA prescribing information. (5): Gall H et al. J Heart Lung Transplant. 2017;36:957-967. (6): Kiely DG et al. Thorax. 2025;80(Suppl 2):A62.1. WHO = World Health Organization; PH = pulmonary hypertension; PH-ILD = pulmonary hypertension associated with interstitial lung disease; PAH = pulmonary arterial hypertension; IPF = idiopathic pulmonary fibrosis; CTD-ILD = connective tissue disease-associated interstitial lung disease; ILD = interstitial lung disease; FDA = U.S. Food and Drug Administration.

Clinical and Pre-Clinical Results Point toward Potential in PH-ILD

- **Clinical evidence:** PROSERA demonstrated favorable results in PAH-CTD patients (placebo-adjusted 6MWD +37.0 m; n=87; nominal p=0.0104), alongside substantial reductions in fibrosis-like parenchymal features and multi-compartment vascular remodeling
- **Human biomarker evidence:** TORREY showed reduced circulating markers associated with extracellular-matrix remodeling, inflammation and fibrosis[1,2]
- **Patient-derived evidence:** Seralutinib reduced pro-collagen Iα1, fibronectin, MMP-3, TIMP-1 and MCP-1 in IPF lung tissue, with no observed cytotoxicity[3]
- **Mechanistic evidence:** Seralutinib inhibited fibroblast activation and modulated disease-relevant fibrotic and inflammatory pathways across IPF lung cell populations[3,4]
- **Functional preclinical evidence:** Seralutinib reduced fibrosis and preserved lung function in an established pulmonary-fibrosis model[5]

IPF = idiopathic pulmonary fibrosis; CTD = connective tissue disease; 6MWD = 6-minute walk distance; MMP-3 = matrix metalloproteinase-3; TIMP-1 = tissue inhibitor of metalloproteinases-1; MCP-1 = monocyte chemoattractant protein-1. (1): Ghofrani HA et al. Am J Respir Crit Care Med. 2024;209:A7383. (2): Osterhout R et al. Eur Respir J. 2024;64(Suppl 68):OA1872. (3): Sitapara R et al. Eur Respir J. 2025;66(Suppl 69):PA1864. (4): Benfeitas R et al. Pulm Circ. 2026;16:e70315:36. (5): Murgo A et al. PFF Summit 2025 poster.

PH-ILD Presents a Significant Market Opportunity

	PAH	PH-ILD
US Prevalence	~50k[1]	~60-100k+[3]
Competitive intensity	17 marketed products	2 marketed products (both treprostinil)
5-year survival rate	57%[2]	23%[4]
Generics	8 generic products	0 generic products

Patients living with PH-ILD are deeply underserved

(1): Institute for Clinical and Economic Review (ICER). Report at a Glance: Pulmonary Arterial Hypertension (PAH). January 2024; prevalence estimates vary widely across sources.
(2): Chang KY et al, J Am Heart Assoc. 2022 May 3;11(9):e024969;
(3): Based on internal company estimates.
(4): Gall H et al, J Heart Lung Transplant 2017;36(9):957-967.

Milestones & Financial Overview

Near Term Milestones

- Sept. 2026: Expect to Submit Seralutinib NDA to FDA for the treatment of PAH
- Nov. 2026: Potential FDA Acceptance of NDA Filing
- 3Q27: Potential Seralutinib FDA Decision

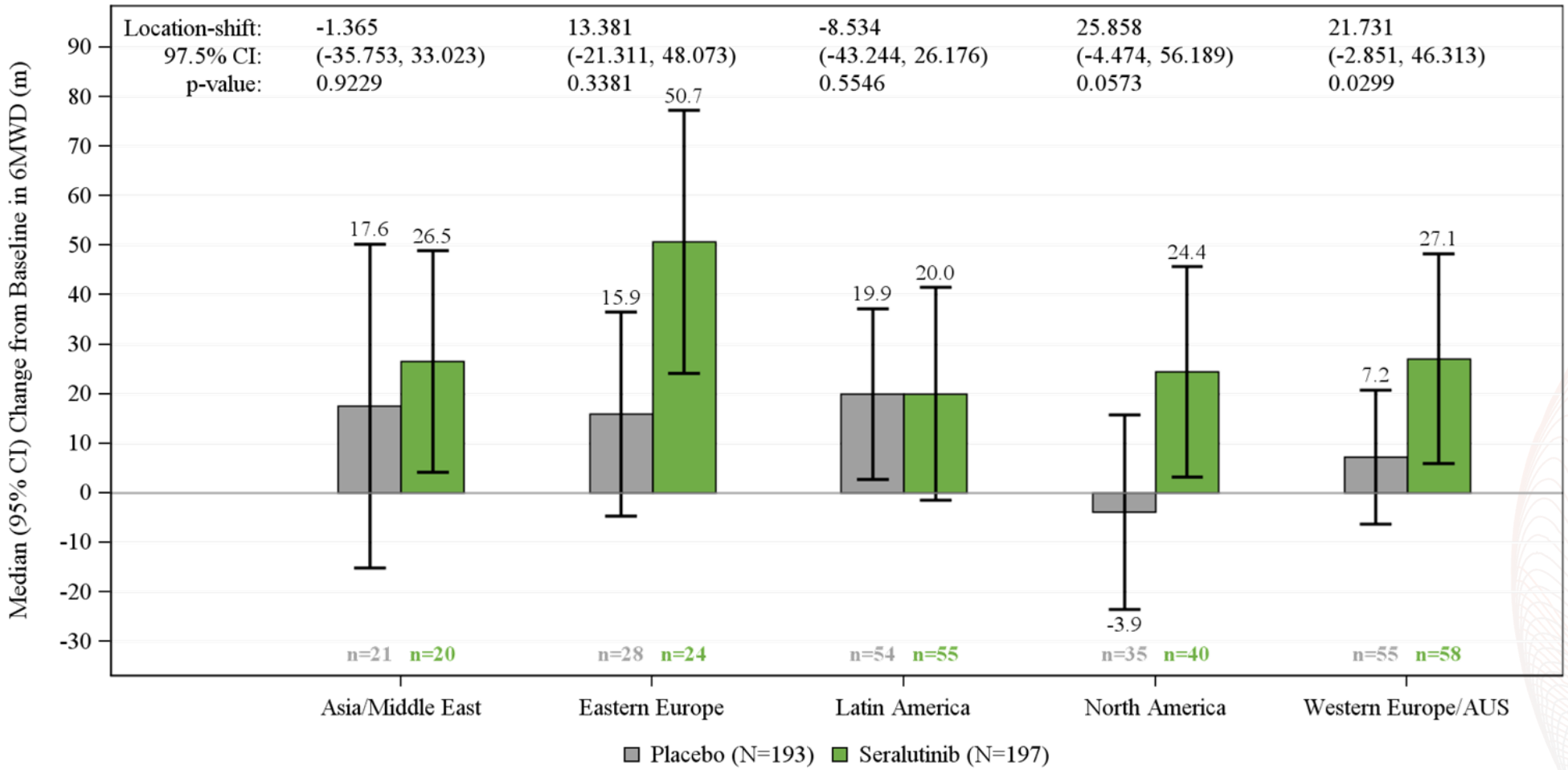
Financial Overview	
Cash, Cash Equivalents and Marketable Securities *(As of 6/30/26)*	~$57mm
Post-quarter: One-time payment from Chiesi	+$5mm
Post-quarter: PIPE initial closing Gross proceeds; does not account for related fees & expenses	+$25mm
Principal of Convertible Notes Outstanding *(After May 2026 exchange; includes ~$18.9mm legacy notes and $65.2mm new secured notes due 2030)*	~$84mm

PIPE announced Aug. 21, 2026: Additional $125mm committed upon FDA acceptance of the seralutinib NDA in PAH.

PIPE = private placement in public equity; mm = millions.

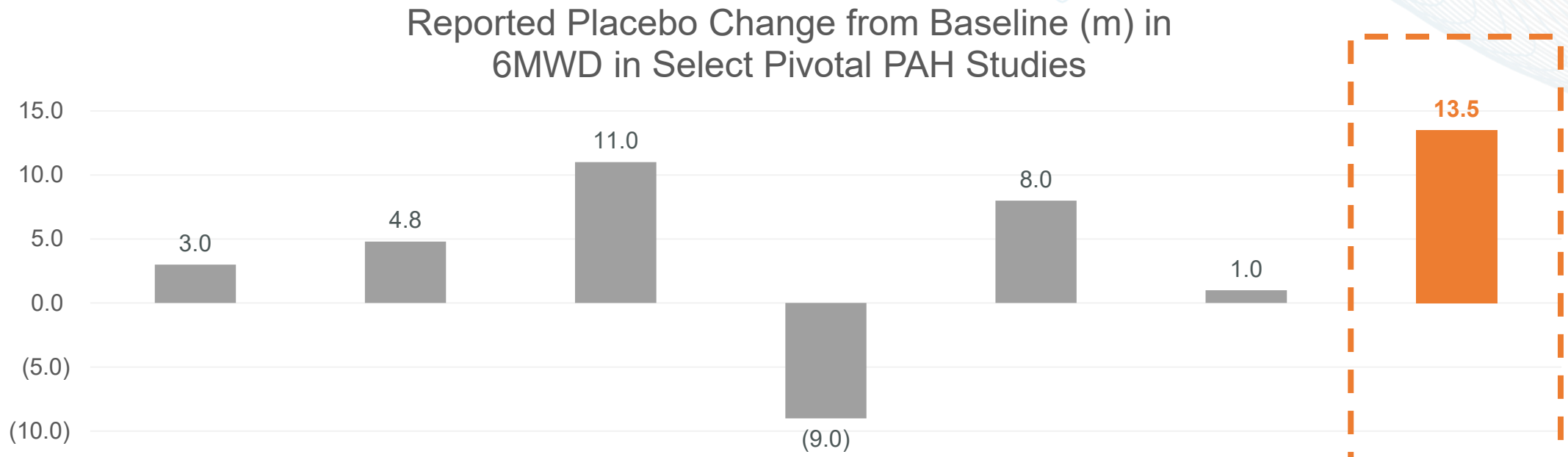
Appendix

Placebo Effect was More Pronounced in Certain Regions, Particularly Latin America & Asia/Middle East



6MWD = 6-minute walk distance; CI = confidence interval.

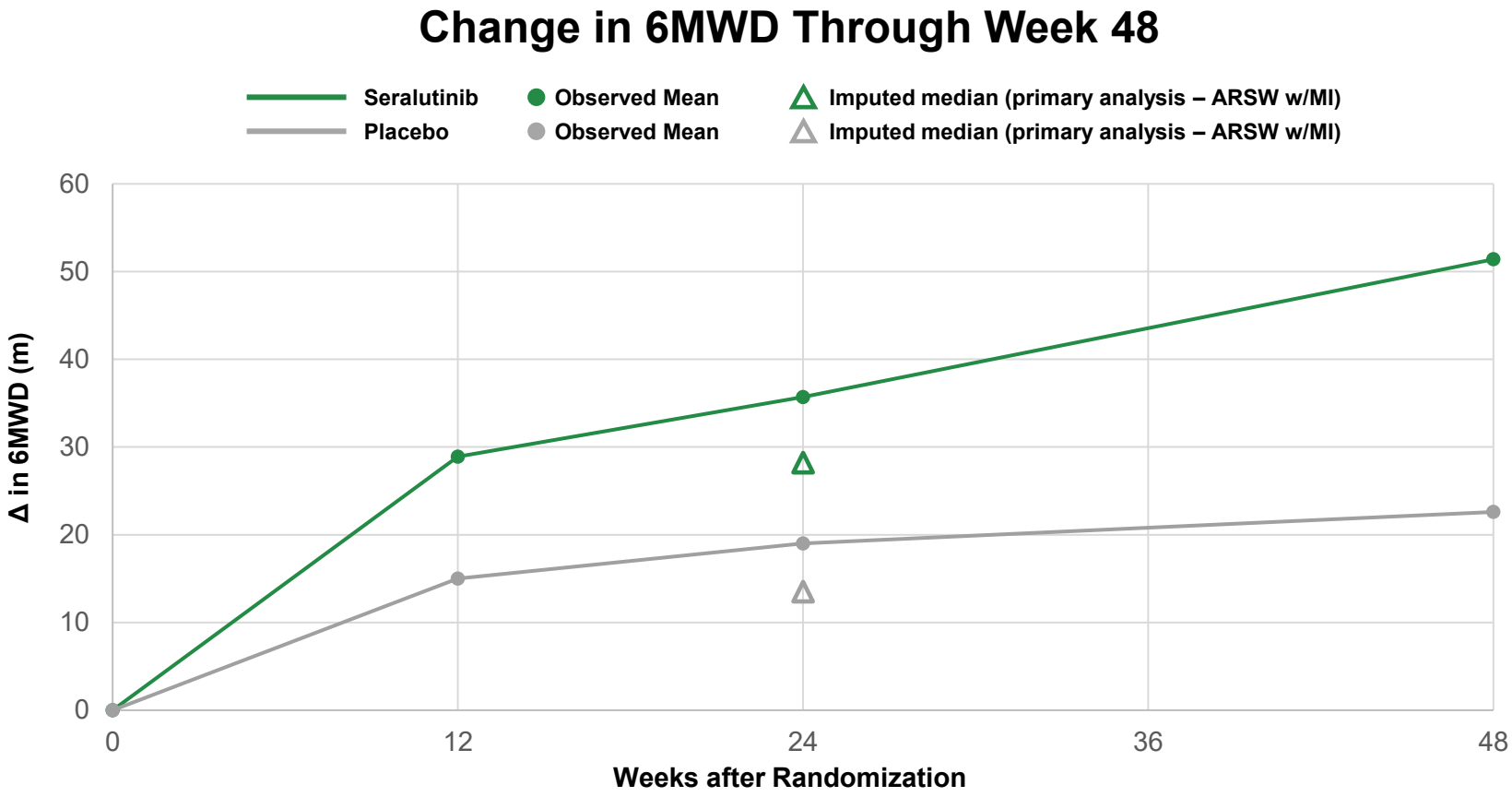
PROSERA Had Elevated Placebo 6MWD Response vs. Other Pivotal Studies For Add-On Treatments in PAH



Compound	Inhaled Tre	Oral Tre	Oral Tre	Selexipag	Oral Tre	Sotatercept	Seralutinib
Study	TRIUMPH[1]	FREEDOM-C[2]	FREEDOM-C2[3]	GRIPHON[4]	FREEDOM-EV[5]	STELLAR[6]	PROSERA
Timepoint	Week 12	Week 16	Week 16	Week 26	Week 24	Week 24	Week 24
Year	2007	2010	2010	2014	2018	2022	2026
Pbo-Adj. Treatment Δ	20m	11m	10m	12m	8m	41m	13m
Background PAH Therapies	PDE5 or ERA	PDE5 + ERA	PDE5 + ERA	PDE5 + ERA	PDE5 + ERA	PDE5 + ERA + Prostacyclin	PDE5 + ERA + Prostacyclin + Sotatercept

Note: Seralutinib is an investigational agent no approved for use in any jurisdiction. The clinical data presented in this presentation comparing seralutinib and other products and product candidates are based on cross-study comparisons and are not based on any head-to-head clinical trials. Differences exist between trial designs, patient characteristics and other factors, and caution should be exercised in drawing any conclusions from a comparison of the data across studies as cross-study comparisons are inherently limited and such data may not be directly comparable. Tre = treprostinil; Pbo = placebo; ERA = endothelin receptor antagonist; PDE5 = phosphodiesterase type 5 inhibitor; 6MWD = 6-minute walk distance. (1): McLaughlin, et al. JACC 2010; (2): clinicaltrials.gov/study/NCT00325442; (3): clinicaltrials.gov/study/NCT00887978; (4): Sitbon, et al. NEJM 2015; (5): White, et al. AJRCCM 2020; (6): Hoeper, et al. NEJM 2023

Observed Mean Change in 6MWD Continued to Separate From Placebo After Week 24 in Sub-Population That Reached Week 48



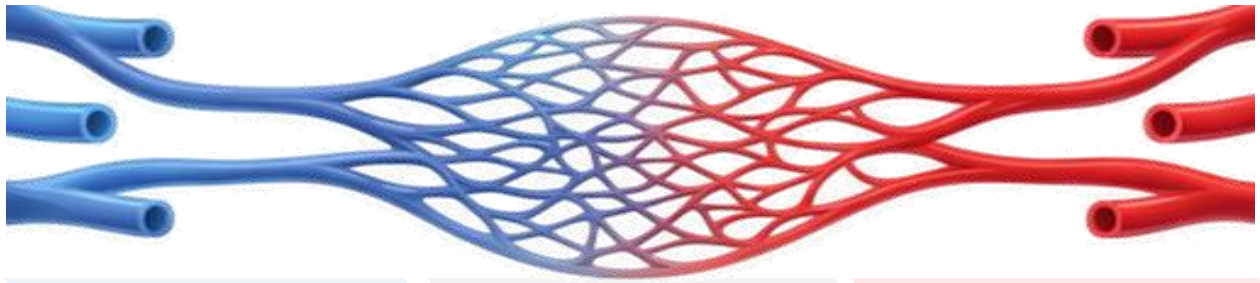
No. Observed				
Seralutinib	197	186	165	57
Placebo	193	186	176	65

Primary Analysis at Week 24:	
H-L Location Shift:	13.298m
97.5% CI:	-0.574, 27.169
p-value:	0.0320

ARSW w/MI = aligned rank stratified Wilcoxon test with multiple imputation; H-L Location Shift = Hodges-Lehmann location-shift estimate (treatment effect vs placebo); CI = confidence interval; 6MWD = 6-minute walk distance.

PAH Is a Multi-Compartment Vascular Disease, Not Just Arterial Remodeling

- PAH is a multi-compartment disease including:[1,2,3]
  - Arterial remodeling
  - Capillary dropout
  - Venous underfilling
  - Perivascular fibrosis
  - Inflammation
- FRI captures sections of an integrated arterial – capillary – venous system and the surrounding parenchymal space[1,4]
  - Changes in one compartment (whether positive or negative) propagate to others[2,3]



Pulmonary Arteries	Capillary Bed	Pulmonary Veins
Deoxygenated Blood Flow	*Gas Exchange*	*Oxygenated Blood Flow*
• PDGFR signaling thickens artery walls and narrows the vessels[3,6] • Reversal of this process is consistent with redistribution from larger to smaller arteries [4,5] • CT FRI measurable[1,4]	• Precapillary arteriolar lesions and parenchymal fibrosis converge on the capillary bed, below CT resolution[2,3] • Damage here is a major driver of clinical decline[2] • Inhaled seralutinib deposits in alveoli and penetrates to capillary bed[5]	• Venous underfilling reflects reduced transpulmonary flow (not venous disease)[1,4] • Volumes are reduced in PAH;[1] consistent with downstream flow impairment • CT FRI measurable[1,4]

Parenchymal Space	• Parenchymal remodeling with fibrotic-like features contributes to vascular stiffening; macrophage infiltration and fibrous tissue buildup drive tissue density changes detectable by CT[2,3,4]

(1): Rahaghi FN et al. Chest. 2021;160:2220-2231. (2): Humbert M et al. Eur Respir J. 2019;53:1801887. (3): Pullamsetti SS et al. Int J Mol Sci. 2023;24:12653. (4): Gossamer Bio. PROSERA Phase 3 CT-FRI Substudy. 2026. Data on file. (5): Galkin A et al. Eur Respir J. 2022;60:2102356; Gossamer Bio, pulmonary deposition data on file. (6): Shimoda LA, Laurie SS. J Mol Med. 2013;91:297-309. CT = computed tomography; FRI = functional respiratory imaging; PAH = pulmonary arterial hypertension; PDGFR = platelet-derived growth factor receptor.

Functional Respiratory Imaging (FRI) Links Vascular Structure to Clinical Trajectory in PAH

- At baseline, arterial, venous, and vascular complexity parameters correlated with pulmonary vascular resistance (PVR), mean pulmonary arterial pressure (mPAP), cardiac output, and clinical outcome measures (N=156), relationships not detectable in TORREY substudy
- Furthermore, changes in FRI parameters correlated with improvements in 6MWD, NT-proBNP, and risk scores, linking structural imaging findings to clinical benefit
- This supports FRI as a biologically and clinically relevant readout in PAH, anchoring the treatment-effect data

Arterial Correlations

Correlations with Baseline Characteristics

+: $p < 0.05$
++: $p < 0.01$
+++: $p < 0.001$

	PVR	MPAP	NT-proBNP	REVEAL Lite 2	ESC/ERS	FEV1	FVC
BV5A/BV10A	+++	+++	++	+	++		
BV5A%	+	++	++	+	++	++	+
BV10A%	++	+++	+		+	+++	

Venous Correlations

Correlations with Baseline Characteristics

+: $p < 0.05$
++: $p < 0.01$
+++: $p < 0.001$

	PVR	CO	FEV1	FVC
TVV	+	++		
BV10V	+++	+++		
BV510V	+	+++	+	+
BV5V	+	+		
FDV		+		

FRI = functional respiratory imaging; PVR = pulmonary vascular resistance; mPAP = mean pulmonary arterial pressure; NT-proBNP = N-terminal pro–B-type natriuretic peptide; REVEAL Lite 2 = Registry to Evaluate Early and Long-Term PAH Disease Management Lite 2 risk score; ESC/ERS = European Society of Cardiology / European Respiratory Society; CO = cardiac output; 6MWD = 6-minute walk distance; FEV1 = forced expiratory volume in 1 second; FVC = forced vital capacity; BV5A = arterial vessel volume <5 mm^2; BV10A = arterial vessel volume >10 mm^2; BV5A% = arterial vessel volume <5 mm^2 as proportion of total blood volume; BV510V = venous vessel volume 5–10 mm^2; BV5V = venous vessel volume <5 mm^2; TVV = total venous blood volume; BV10V = venous vessel volume >10 mm^2; FDV = fractal dimension of venous vessels.
Rahaghi et al. Chest 2021;160(6):2220-2231
All correlations at baseline, pooled across treatment arms.