gossamerbio®

1Q26 Earnings Update

May 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 our future results of operations and financial position, business strategies and plans, research and development plans, the anticipated timing, costs, design and conduct of our ongoing and potential clinical trials and preclinical studies for seralutinib, the timing and likelihood of regulatory filings and approvals for seralutinib, including our plans to meet with the FDA and file an NDA, our ability to commercialize seralutinib, our plans to consummate the exchange offer and the anticipated benefits therefrom, and the timing and likelihood of success, plans and objectives of management for future operations, are forward-looking statements.

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: topline results we report are based on preliminary analysis of key efficacy and safety data, and such data may change following a more comprehensive review of the data related to the clinical trial and such topline data may not accurately reflect the complete results of a clinical trial; we may not be able to identify a development path forward for seralutinib or submit an NDA on the timeframe we expect or at all, whether as a result of FDA feedback or otherwise, and any path forward may require additional capital and other resources, which may not be available on reasonable terms, if at all, or may limit the commercial opportunity for seralutinib; we may need to further evaluate our current workforce in light of potential development paths for seralutinib; potential delays in the commencement, enrollment and completion of clinical trials; comparative efficacy and safety information is not based on a head-to-head comparison and differences exist between study designs and subject characteristics which could confound the results; our 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 any future clinical trials and preclinical studies for seralutinib; regulatory developments in the United States and foreign countries; unexpected 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; our ability to obtain and maintain intellectual property protection for seralutinib; our ability to comply with its obligations in collaboration agreements with third parties or the agreements under which we license intellectual property rights from third parties; we may use our capital resources sooner than we expect; unstable market and economic conditions and changes in healthcare legislation, tariffs and trade policies may adversely affect our business and financial condition and the broader economy and biotechnology industry; we may not be able to complete the exchange offer on the anticipated timeline or at all and we may not realize the anticipated benefits therefrom; and other risks described in our prior filings with the Securities and Exchange Commission (SEC), including under the heading “Risk Factors” in our annual report on Form 10-K for the year ended December 31, 2025 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.

This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk. These and other factors could cause results to differ materially from those expressed in the estimates made by the independent parties and by us.

The offering, issuance and sale of the new convertible senior notes as part of the exchange offer has not been registered under the Securities Act of 1933, as amended, or any other securities laws. This presentation shall not constitute an offer to sell, or the solicitation of an offer to buy, the new convertible notes, shares of common stock (or prefunded warrants) and purchase warrants offered in the exchange offer, the shares of common stock issuable upon conversion of the convertible notes, prefunded warrants or purchase warrants, the existing convertible notes or any other securities, nor will there be any sale of such securities or any other securities, in any state or other jurisdiction in which such offer, sale or solicitation would be unlawful.

Speakers on Today's Call

- **Faheem Hasnain**, Co-Founder, Chairman & Chief Executive Officer
- **Bryan Giraudo**, Chief Operating Officer & Chief Financial Officer
- **Bob Smith**, Chief Commercial Officer
- **Caryn Peterson**, Executive Vice President, Regulatory Affairs
- **Dr. Rob Roscigno**, Senior Vice President, Clinical Development, Pulmonary Vascular Disease
- **Dr. Jean-Marie Bruey**, Senior Vice President, Translational Medicine and Research
- **Dr. Rainer Zimmermann**, Vice President, Medical Affairs
- **Dr. Megan Flynn,** Vice President, Medical Affairs
- **Dr. Robin Osterhout**, Executive Director, Translational Medicine

I. Regulatory Update

In-Person FDA Type B Meeting Confirmed for Mid-June

- FDA has confirmed an in-person meeting for Mid-June
- Meeting will address the regulatory path forward for seralutinib, including discussion of the totality of clinical evidence across the clinical program
- Subject to a productive Type B meeting outcome, Gossamer is targeting an NDA submission in September 2026
- We expect to submit an NDA under the basis of one adequate and well-controlled clinical investigation (PROSERA) plus confirmatory evidence (TORREY), supported by the seriousness of PAH, high unmet need, and seralutinib's novel mechanism of action

II. PROSERA CT FRI Substudy Topline Results

PROSERA CT FRI Substudy:
Most Comprehensive Controlled Vascular Imaging Dataset in PAH to Date

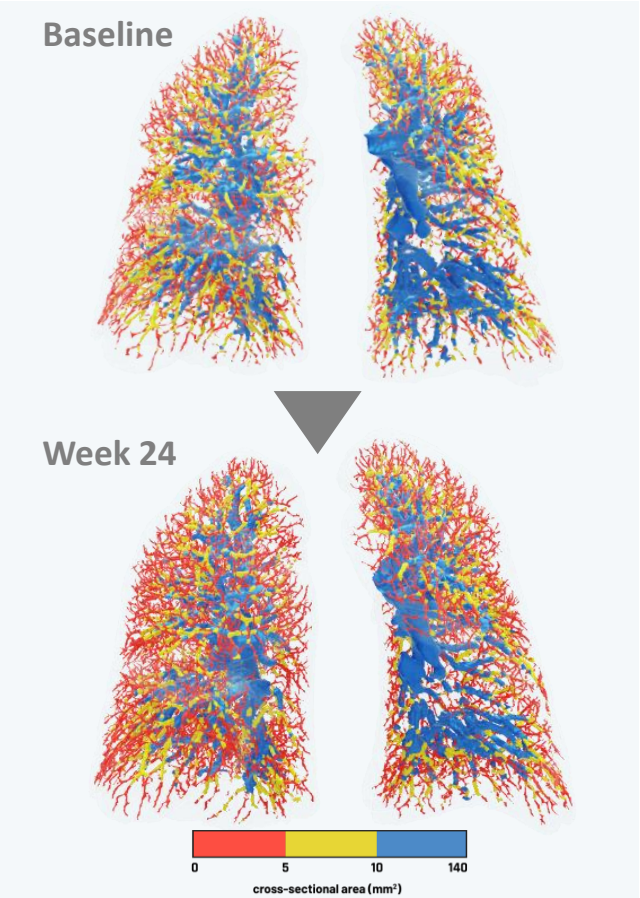
- Clinical endpoints show whether patients improved; computed tomography functional respiratory imaging (CT FRI) reveals the anatomical basis of that improvement, which is key for a non-vasodilator
- Built on seralutinib's initial arterial reverse remodeling signal seen in Phase 2 TORREY CT FRI substudy (n=19); the PROSERA CT FRI substudy was designed to further validate this treatment effect
- We believe this to be the largest and most comprehensive CT FRI dataset from a controlled therapeutic trial in PH: 162 enrolled, 125 paired scans at Week 24, in patients on multiple concurrent background PAH vasodilators
- Prespecified exploratory substudy using an updated algorithm with improved proximal large-vessel capture versus TORREY
- Substudy was representative of the broader PROSERA intent-to-treat (ITT) (n=390) on demographics, hemodynamics, and risk profile
- Week 24 clinical endpoints in the substudy were consistent with the broader ITT population

FRI = functional respiratory imaging; CT = computed tomography; 6MWD = 6-minute walk distance; NT-proBNP = N-terminal pro–B-type natriuretic peptide; ITT = intent-to-treat; REVEAL Lite 2 = Registry to Evaluate Early and Long-Term PAH Disease Management Lite 2 risk score.

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



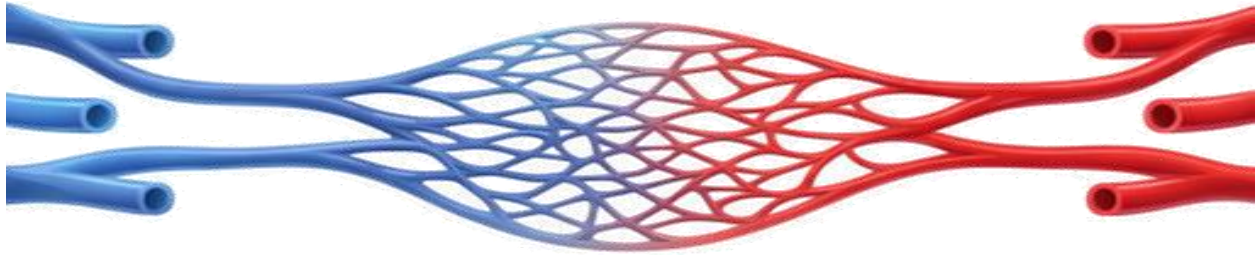
Color indicates vessel cross sectional area. Red denotes small-caliber vessels; blue denotes large-caliber vessels. Redistribution from blue to red is consistent with reverse remodeling. Individual result; treatment effects vary by patient.

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.

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

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

Pulmonary Arteries	Capillary Bed	Pulmonary Veins



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

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

CT = computed tomography; FRI = functional respiratory imaging; PDGFR = platelet-derived growth factor receptor.

1) Rahaghi FN et al. Chest 2021;160(6):2220-2231

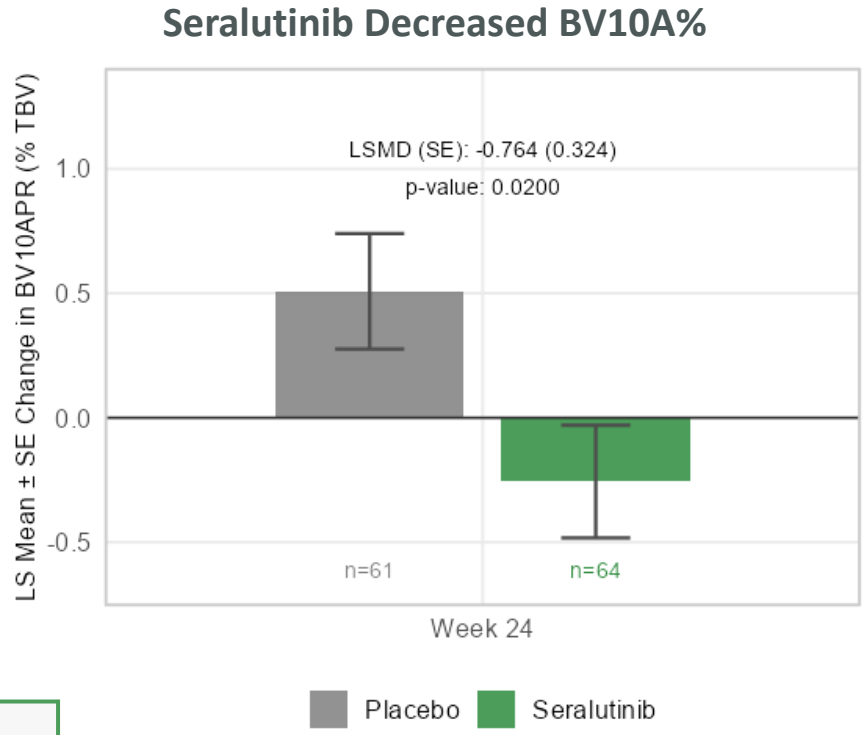
Seralutinib is Designed to Act Across Arteries, Veins, and Lung Parenchyma

	Pulmonary Arteries	Parenchymal Space	Pulmonary Veins
PAH & Seralutinib	PDGFRα/β → PASMC proliferation → Medial hypertrophy → Seralutinib inhibits PDGFRα/β → Redistribution from large arteries to small arteries	PDGFRα/β (fibroblasts) + CSF1R (macrophages) + c-KIT (mast cells) → Fibroblast activation, inflammation, and parenchymal remodeling → Seralutinib inhibits PDGFRα/β + CSF1R + c-KIT → Decreased fibrosis-like parenchymal features	Upstream arterial remodeling + anti-fibrotic effects (incl. sub-CT capillary bed) → Improved transpulmonary flow → Venous recovery
Seralutinib CT FRI Treatment Effect in PROSERA v. Placebo	**Large arterial blood volume proportion ↓** (BV10A%, p=0.020)	**Fibrosis-like parenchymal volume ↓** (FV, p=0.026) **Normalized fibrosis-like volume↓** (FVN, p=0.025)	**Total venous volume ↑** (TVV, p=0.016) **Small venous blood volume ↑** (BV5V, p=0.034) (BV510V, p=0.041) **Venous fractal dimension ↑** (FDOPV, p=0.044)

CT = computed tomography; FRI = functional respiratory imaging; PDGFRα/β = platelet-derived growth factor receptor alpha/beta; PASMC = pulmonary artery smooth muscle cell; CSF1R = colony stimulating factor 1 receptor; BV10A% = arterial vessel volume >10 mm^2 as proportion of total blood volume; FV = fibrosis volume; FVN = normalized fibrosis volume (FV/lung volume); TVV = total venous blood volume; BV5V = venous vessel volume <5 mm^2; BV510V = venous vessel volume 5–10 mm^2; FDOPV = fractal dimension of pulmonary venous vessels; IPF = idiopathic pulmonary fibrosis; HAA = High Attenuation Area. All p-values are nominal and unadjusted for multiplicity.
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. Methodologically analogous to the High Attenuation Area (HAA)

Seralutinib Showed Reversed Proximal Arterial Remodeling

- Seralutinib significantly decreased large arterial blood volume proportion (BV10A%) v. placebo, indicating of proximal arterial decompression
- Consistent with TORREY arterial results, now reproduced in a much larger population
- BV10A% demonstrated among the strongest clinical correlations of any FRI parameter
- Changes at 24 weeks correlated with improvements across multiple clinical endpoints



BV10A% Clinical Correlations (p<0.05)

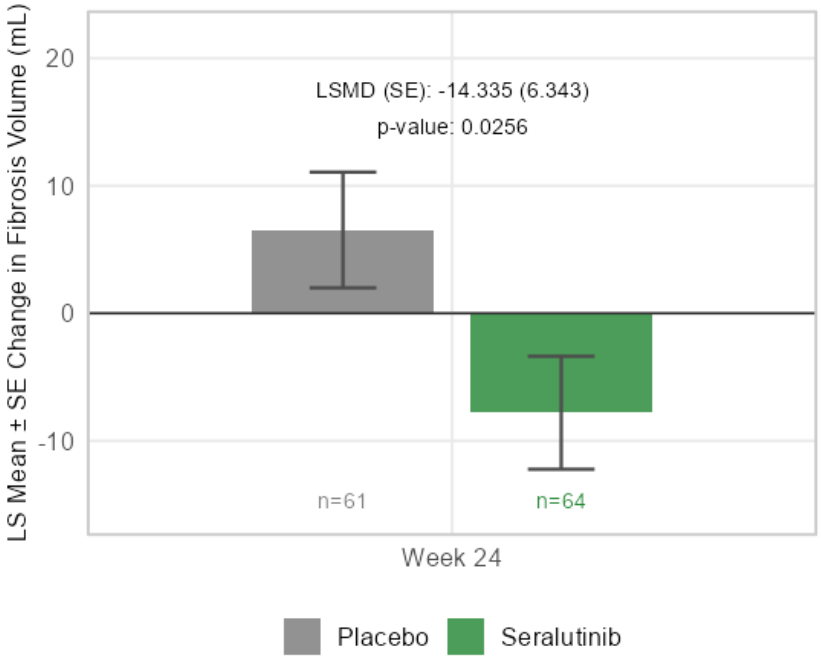
- **At baseline:** correlated with PVR, mPAP, NT-proBNP, ESC/ERS risk
- **Change at 24 Weeks:** correlated with 6MWD, NT-proBNP, REVEAL Lite 2, ESC/ERS risk

BV10A% = arterial vessel volume >10 mm² as proportion of total blood volume; 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; ESC/ERS = European Society of Cardiology / European Respiratory Society risk strata; PVR = pulmonary vascular resistance; mPAP = mean pulmonary arterial pressure; TORREY = Phase 2 trial of seralutinib in PAH.
Rahaghi FN et al. *Chest* 2021;160(6):2220-2231. All p-values are nominal and unadjusted for multiplicity.

Seralutinib Reduces Fibrosis-Like Parenchymal Features While Placebo Increases

- Seralutinib significantly decreased functional respiratory imaging (FRI) fibrosis metrics [(1)], placebo increased
- First demonstration of statistically significant fibrotic reduction in controlled PAH trial
- Decreases seen across subgroups, including non-connective tissue disease (CTD) patients
- Reduction in fibrosis-like parenchymal features is consistent with PDGFR, CSF1R and c-KIT pathway inhibition, an antifibrotic effect distinct from vasodilation
- The same fibrotic and inflammatory pathobiology drives PH-ILD and other fibrotic lung diseases, supporting the potential relevance of seralutinib beyond PAH

Seralutinib Decreased Fibrosis-like Volume Metric



Fibrotic Metrics' Clinical Correlations (p<0.05)

- **At baseline:** correlated with NT-proBNP, ESC/ERS risk
- **Change at 24 Weeks:** correlated with NT-proBNP, REVEAL Lite 2

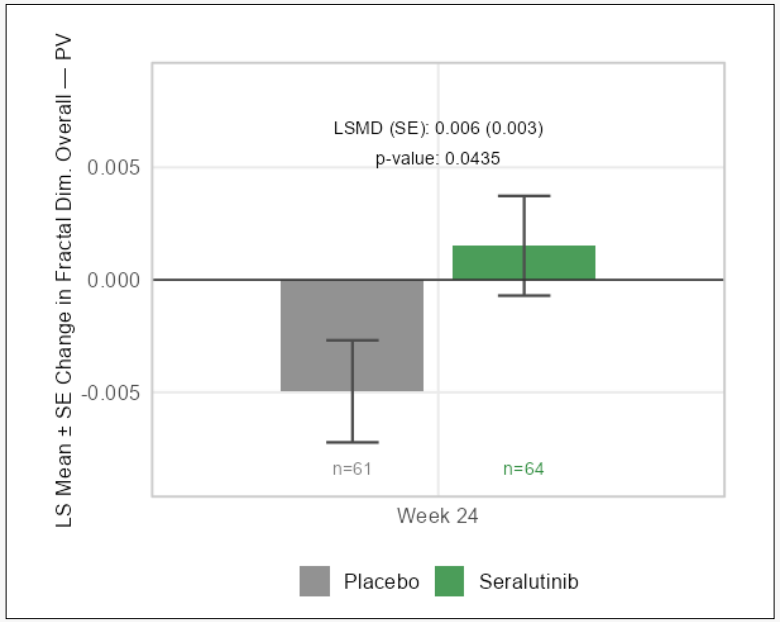
FRI = functional respiratory imaging; FV = fibrosis volume; CT = computed tomography; NT-proBNP = N-terminal pro–B-type natriuretic peptide; ILD = interstitial lung disease; IPF = idiopathic pulmonary fibrosis; CTD = connective tissue disease; FVN = normalized fibrosis volume; ESC/ERS = European Society of Cardiology / European Respiratory Society; REVEAL Lite 2 = Registry to Evaluate Early and Long-Term PAH Disease Management Lite 2 risk score; CSF1R = colony stimulating factor 1 receptor; c-KIT = KIT proto-oncogene receptor tyrosine kinase; PH-ILD = pulmonary hypertension–interstitial lung disease.

(1) FRI fibrosis volume (FV) quantifies CT voxel-level features characteristic of fibrotic ILD (e.g., reticulation, ground-glass), derived from FibroNet deep-learning algorithm trained on confirmed IPF patient datasets.

(2) Osterhout et al, AJRCCM, *Accepted* (2026)

First Evidence of Venous Vascular Recovery in a Controlled PAH Trial

- In the PROSERA substudy, seralutinib significantly increased venous blood volume; placebo decreased
- Consistent increases across venous vessel sizes and vascular branching (fractal dimension), suggesting improved microvascular flow and perfusion
- Venous volumes and vascular branching correlated with PVR and cardiac output at baseline ($p<0.05$, Spearman)
- Venous improvements likely capture the totality of upstream treatment effects, including arterial, anti-fibrotic, and sub-CT capillary bed changes
- First demonstration of venous vascular recovery in a controlled PAH trial



Venous Fractal Dimension (FDOPV; p=0.0435)

- Increased vascular branching
- Increased venous branching complexity suggests structural vascular recovery, not merely passive volume redistribution
- Consistent with attenuation of vascular pruning, a hallmark of progressive PAH[1]

Venous Clinical Correlations ($p<0.05$)

- **At baseline:** venous volumes correlated with PVR and cardiac output; BV510V also correlated with FEV1 and FVC; FDOPV correlated with cardiac output
- **Change at 24 Weeks:** BV10V correlated with REVEAL Lite 2; FDOPV correlated with FEV1

TVV = total venous blood volume; BV5V = venous vessel volume <5 mm^2; BV510V = venous vessel volume 5–10 mm^2; BV10V = venous vessel volume >10 mm^2; FDOPV = fractal dimension of pulmonary venous vessels; CT = computed tomography; FRI = functional respiratory imaging; PVR = pulmonary vascular resistance; FEV1 = forced expiratory volume in 1 second; FVC = forced vital capacity.

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.

1) Rahaghi et al. *Chest* 2021;160(6):2220-2231

All correlations at baseline, pooled across treatment arms.

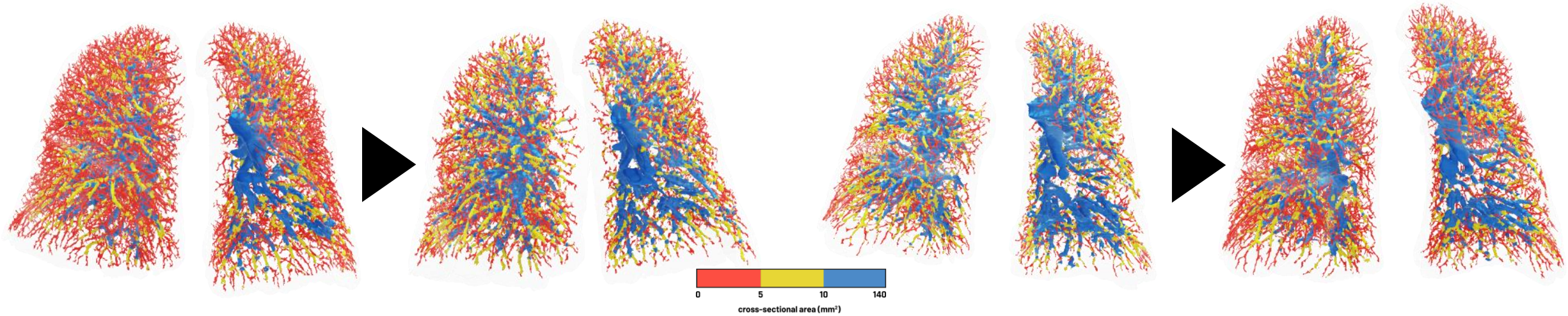
Seralutinib Demonstrated Statistically Significant Treatment Effects Across Multiple Vascular Compartments

Category	Parameter	Abbrev.	Effect	p-value	Interpretation
Arterial	Arterial vessel volume >10mm² / TBV	BV10A%	↓ Decrease	0.020*	Reduced proportion of large-artery volume consistent with proximal decompression
Arterial	Arterial vessel volume >10mm²	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 <5mm²	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 <5mm²	BV5V	↑ Increase	0.034*	Increased small venous vessel volume
Venous	Venous vessel volume 5–10mm²	BV510V	↑ Increase	0.041*	Increased mid-size venous vessel volume
Venous	Venous vessel volume >10mm²	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 <5mm² / total blood volume; SAP = statistical analysis plan; FRI = functional respiratory imaging; CT = computed tomography. In addition to the 7 nominally statistically significant prespecified parameters shown above, BV5VPR (venous vessel volume <5mm² / 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)

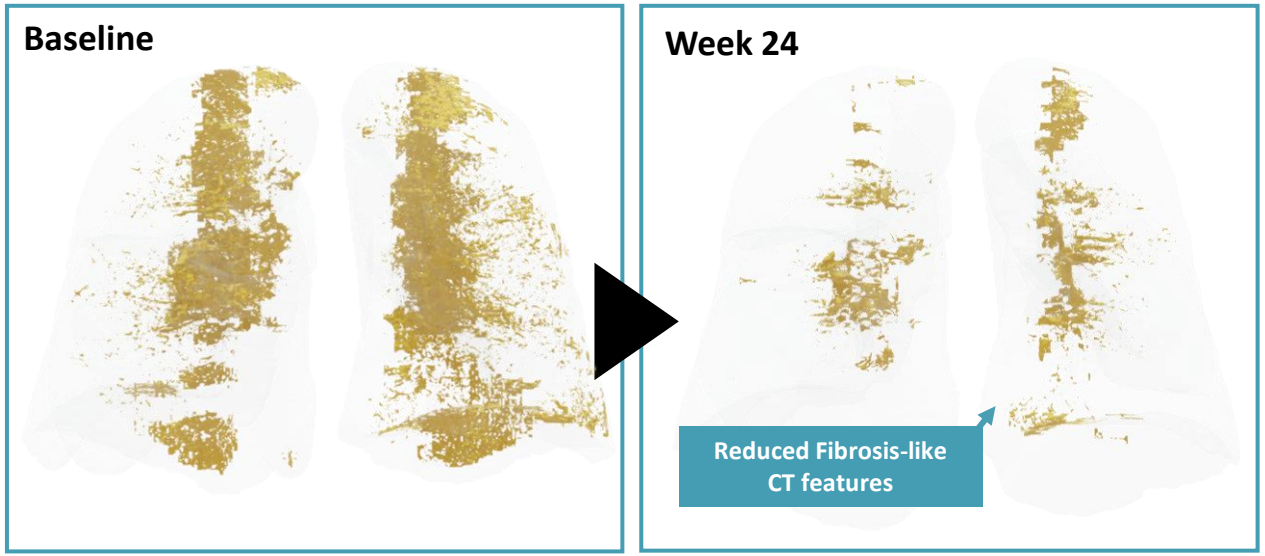
Placebo patient		Seralutinib patient	
Baseline	**Week 24**	**Baseline**	**Week 24**



60y Female, PAH-CTD, FC 3, Triple Therapy		*70y Female, IPAH, FC 3, Triple Therapy*	
Δ 6MWD	-1 m	Δ 6MWD	+55 m
Δ NT-proBNP	-143 ng/L	Δ NT-proBNP	-324 ng/L
Δ BV10A, BV5A	+12.6 mL, -6.8 mL	Δ BV10A, BV5A	-4.2 mL, +5.5 mL
Δ Total Venous Volume	-8.9 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 mm²; BV5A = arterial vessel volume <5 mm².
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.

Why Does It Matter?

- Multi-compartment vascular and parenchymal remodeling effects of this breadth have not been shown by traditional vasodilator therapies
- First controlled-trial imaging evidence of arterial decompression with concomitant venous vascular expansion, indicating coordinated restoration of pulmonary vascular flow
- Imaging and clinical results point to a mechanism-based effect on PAH disease biology, not just symptomatic relief, consistent with PDGFRα/β + CSF1R + c-KIT pathway inhibition
- Imaging results correlated with improvements in 6MWD, NT-proBNP, and REVEAL Lite 2, linking structural remodeling to clinical benefit
- Seralutinib's mechanism of action may extend to fibrotic lung diseases, particularly those with a pulmonary vascular component
- These data strengthen the cumulative weight of evidence for seralutinib, extending the consistent signal seen across the program's clinical trials: the placebo-controlled Phase 1b, the Phase 2 TORREY Study, and the Phase 3 PROSERA Study

PDGFRα/β = platelet-derived growth factor receptor alpha/beta; CSF1R = colony stimulating factor 1 receptor; c-KIT = KIT proto-oncogene receptor tyrosine kinase; 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. Note: all significance calculations are nominal and unadjusted for multiplicity.

III. Bond Exchange

Addressing the Capital Structure

Existing Notes	$200mm, 5.00% Convertible Senior Notes due 2027
Exchange Consideration	Per $1,000 principal: up to 1,588.2353 shares of common stock (or pre-funded warrants in lieu thereof) + $360 principal amount of new secured notes + 750 early tender purchase warrants
New Notes	7.50% Senior Secured First Lien Convertible Notes due 2030
Debt Reduction(1)	$200mm → $72mm (would reduce debt by $128mm, or 64%)
Maturity Extension(1)	2027 → 2030
Strategic Rationale	Addresses near term capital structure overhang
Noteholder Support	75% of existing noteholders have entered into a Transaction Support Agreement

(1) Assumes 100% participation.

Thank you