

Corporate Presentation

November 2024

Forward Looking Statements

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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. These statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. These known risks and uncertainties are described in detail in our filings with the Securities and Exchange Commission (the "SEC") from time to time. Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur and actual results could differ materially from those projected in the forward-looking statements. Except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise. 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 and we undertake no obligation to revise or update this presentation to reflect events or circumstances after the date hereof.

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I. Overview of Seralutinib

A Novel Investigational Inhaled Treatment for Pulmonary Hypertension (PH)

Gossamer Bio Quick Facts









INVESTIGATIONAL PROGRAM	CLASS (Route of Admin.)	INDICATION	PHASE 1	PHASE 2	PHASE 3	COMMERCIAL RIGHTS
	Tyrosine Kinase Inhibitor (PDGFR,	Pulmonary Arterial Hypertension (PAH)	Registrational Pha Completed Phase 3 Met Primary Endpoint Well-Tolerated	se 3 Study Ongoing 2 Study		Partnered with Chiesi Gossamer leads global development and US commercial efforts in PAH and PH-ILD.
Seralutinib (GB002)	CSF1R, c-KIT) (Inhaled)	Pulmonary Hypertension Associated with Interstitial Lung Disease (PH-ILD)	Registrational Pha to Commence Mid	•		Gossamer is entitled to a 50:50 profit / loss split in the US, exUS royalties and milestones. Chiesi is responsible for 50% of development costs, outside of the ongoing PAH Phase 3.



Seralutinib Overview

A Novel Investigational Inhaled Treatment for PH

- Inhaled kinase inhibitor, designed from scratch to address the underlying disease pathophysiology of pulmonary hypertension (PH)
 - PH is a class of rare lung diseases including PAH and PH-ILD, amongst others
- Status in PAH: Ongoing global, registrational Phase 3 study
- Status in PH-ILD: Expect to initiate a global, registrational Phase 3 mid-2025
- In the positive Phase 2 TORREY Study in PAH patients, seralutinib demonstrated statistically significant¹:
 - Improvement in pulmonary vascular resistance ("PVR", primary endpoint)
 - ✓ Improvement in NT-proBNP, a biomarker of right heart failure
 - ✓ Improvements in right heart structure & function
- In an open-label extension study, seralutinib showed a continued improvement in PVR, with a near doubling of improvement from Week 24 to Week 72²
- Seralutinib has been generally well tolerated to date
- Patent protection to 2039³; Orphan Drug Designation from FDA and EMA





²⁾ diaboth 6, data. An in New 2014, 2015 All Part (2014) Control of the American Security Control o

II. Seralutinib in PAH

PAH is a Rare, Progressive & Fatal Disease



- PAH is a rare, progressive cardiopulmonary disease, with no known cure, affects approximately 50,000 patients in the US¹
- Characterized by high blood pressure in the blood vessels carrying deoxygenated blood from the right side of the heart to the lungs
- Caused when the small arteries in the lungs become narrowed, thickened and / or stiff as a result of pathological remodeling and vasoconstriction
- Heart works harder to pump blood to the lungs, potentially leading to right heart failure and death

Pulmonary Vascular Remodeling: A Key Structural Alteration in PAH

Pathological mechanisms

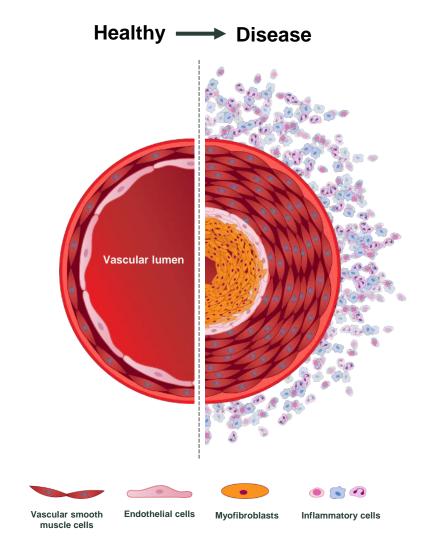


Vascular remodeling of the small pulmonary arteries

- Peri-vascular inflammation
- Neointimal proliferation of endothelial cells and myofibroblasts
- Proliferation and hypertrophy of pulmonary artery smooth muscle cells
- Perivascular fibrosis

Leading to

- Increased pulmonary vascular resistance
- Decreased pulmonary artery compliance
- Right ventricular hypertrophy and right heart failure

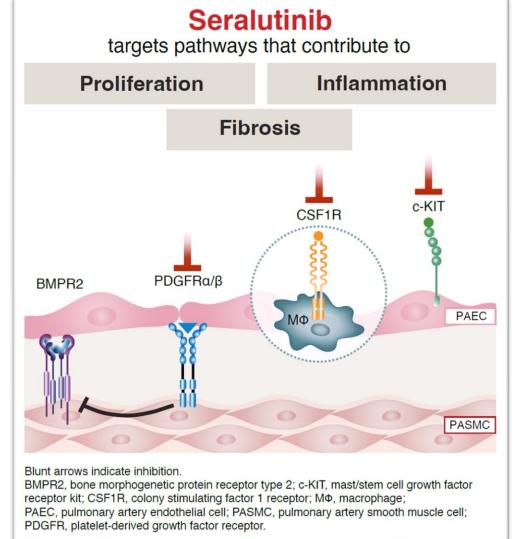






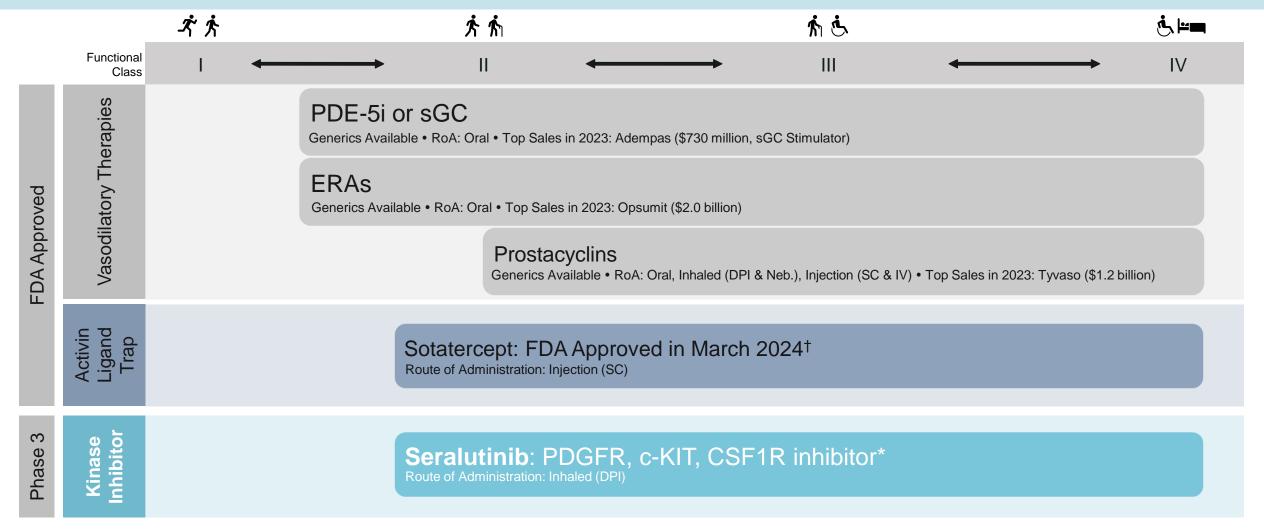
Seralutinib: An Intentional Approach to PAH

- Designed from scratch to address the underlying disease pathophysiology of pulmonary hypertension (PH)
 - Only tyrosine kinase inhibitor (TKI) intentionally developed as an inhaled treatment for PAH
- Inhibiting the PDGFR pathway reverses pulmonary vascular remodeling in animal models of PAH^{1,2}
- TKIs have been used to target this pathway, but safety concerns occurred with oral imatinib (anti-cancer therapy)³
- This led to the development of seralutinib, a distinct nextgeneration TKI to address these concerns
- In preclinical models, seralutinib has shown greater potency and selectivity as compared to imatinib, targeting PDGFRα/ß, CSF1R, and c-KIT, thereby acting on inflammatory, proliferative, and fibrotic drivers of pulmonary vascular remodeling⁴



Hoeper et al. Circulation 2013: 127(10):1128-38.

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



Branded PH drug sales totaled over \$7 billion in sales in 2023.

Sources: Company financial reports.

DPI = dry powder inhaler; Neb. = nebulized; SC = subcutaneous injection; IV = intravenous injection.

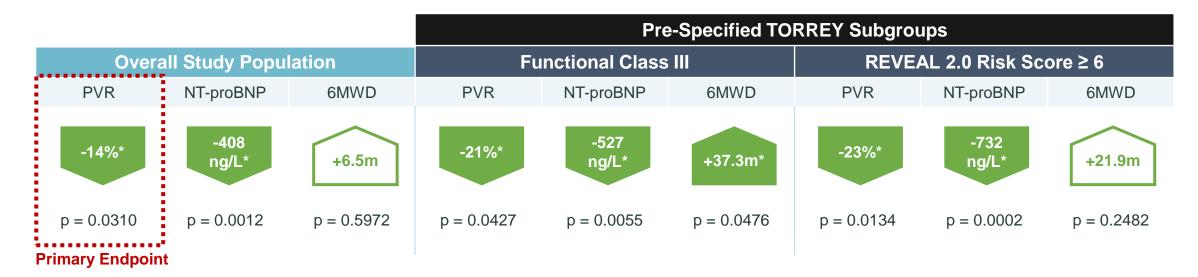
[†]Sotatercept positioning is conjectural and subject to adjustment, given its recent entry into the market and the PAH treatment paradigm.

^{*}Reflects potential positioning for an investigational therapy that is not yet approved and is subject to regulatory review and approval. Safety and efficacy have not yet been established. Subject to change

III. Phase 2 TORREY Study in PAH

TORREY Study Phase 2 Topline Results (24 Weeks)

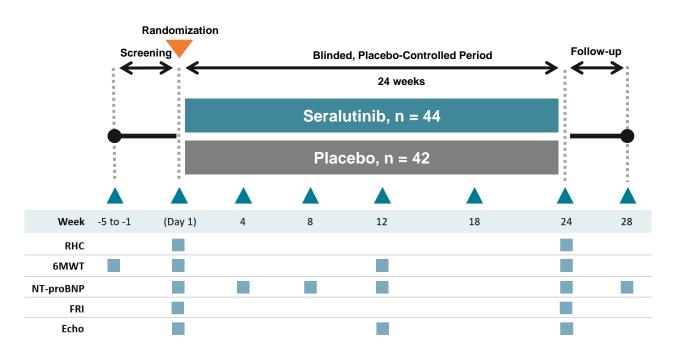
- Met Primary Endpoint: Statistically significant reduction in PVR in heavily-treated study population
- Consistent numerical PVR (primary endpoint) benefit seen in all 21 pre-specified subgroups in favor of seralutinib - enhanced benefit seen in patients with more severe disease at baseline§



- Additional evidence of seralutinib benefit seen in multiple pre-specified endpoints
- Well-tolerated, avoiding side effect profile associated with systemic imatinib in PAH



TORREY: Completed Phase 2 Study of Seralutinib in Patients With Functional Class II and III PAH





Patient Population	Stable FC II & III PAH patients on background therapy, including double & triple therapy
Endpoints	Primary: ∆PVR at Week 24 Key Secondary: ∆6MWD at Week 24 [†] Exploratory: Includes NT-proBNP, Echo
Dosing Regimen	Titrated up to 90mg BID (Started at 60mg BID; protocol allowed for down-titration to 45mg BID)

RHC = right heart catheterization; 6MWT = six-minute walk test; FRI = functional respiratory imagining; Echo = echocardiogram; FC = Functional Class; OLE = open label extension; PVR = pulmonary vascular resistance; 6MWD = six-minute walk distance; BID = twice-daily dosing. Frantz RP, et al. Lancet Respir Med 2024;12(7):523-534.

Selected Baseline Disease Characteristics

(ITT Population)

- Baseline patient PAH disease characteristics were milder than precedent PAH clinical trial populations, in large part due to patient availability during the COVID pandemic, which was ongoing during enrollment
- Despite this, seralutinib demonstrated a statistically significant treatment effect in its primary endpoint
- The TORREY Study is the most well-controlled PAH clinical trial patient population to meet its primary endpoint*

Characteristic	Placebo (N=42)	Seralutinib (N=44)	Total (N=86)	
Number of PAH background therapies – n (%)				
1	2 (4.8)	1 (2.3)	3 (3.5)	
2	16 (38.1)	18 (40.9)	34 (39.5)	Heavily pre-treated patient population
3	24 (57.1)	25 (56.8)	49 (57.0)	
WHO FC – n (%)				Hit Primary Endpoint
Class II	20 (47.6)	30 (68.2)	50 (58.1)	Despite FC Imbalanc
Class III	22 (52.4)	14 (31.8)	36 (41.9)	in Drug & Pbo Arms
PVR (dyne*s/cm ⁵) – mean (SD)	661.3 (164.91)	675.8 (240.35)	668.7 (205.90)	Mildest baseline PAH disease to see
6MWD (m) - mean (SD)	407.1 (107.02)	408.6 (75.11)	407.9 (91.54)	treatment effect*
NT-proBNP (ng/L) - mean (SD)	645.6 (1158.75)	611.0 (714.58)	628.3 (956.83)	
			STELLAR Trial Phase 3 NT-p mean baseline was 1,121.1 PVR was 763.7 dyne*s/cn	ng/L;



Seralutinib Phase 2 TORREY Study Scorecard

Consistently Demonstrated Improvement across Multiple Meaningful Measures

Up to 24 Weeks...

Pre-Specified Endpoint		Assessed Attribute	Measure	Measurement Procedure	Stat. Sig. Improvement*?	p-value	Notes	Citation
Primary	<u> </u>	Pulmonary Resistance & Function	PVR	RHC	/	0.0310	 Demonstrated numerical improvement across all 21 pre-specified subgroups with nominal statistical significance in 7 of them Generally, enhanced benefit seen in higher risk patients 	1
Secondary	序	Exercise Capacity	6MWD	6MWT	#	0.0476#	 Demonstrated stat. sig. improvement in pre-specified subgroup, Functional Class III patients (n = 36)# +37.3-meter improvement v. placebo 	1
Exploratory	•	Heart Failure Biomarker	NT-proBNP	Blood Sample	/	0.0012	 Demonstrated stat. sig. improvement at weeks 12 & 24 Numerical improvement relative to pbo seen at wks 4 & 8 (p < 0.07) 	1
Exploratory		Hemodynamics, Heart Structure, etc.	Multiple	RHC, ECHO		Multiple < 0.05	 Demonstrated stat. sig. improvement in Right Atrium Area, RV Free Wall Strain, PA Compliance, RV Systolic Pressure, PA Systolic Pressure, PA Diastolic Pressure, mPAP 	2
Sub-Study		Lung Blood Vessel Structure & Remodeling	FRI	CT Scan	/	0.028	 Demonstrated stat. sig improvement in imaging-based biomarker of pulmonary vascular pruning, which correlated with improvement in hemodynamics 	3

24 Weeks & Beyond...

Cate	gory	Notes:	Citation
*	Safety & Tolerability	 Generally well-tolerated in the 24-week blinded study and the ongoing OLE (to date) Vast majority of patients were able to reach and maintain highest tested dose, 90mg twice daily Top adverse events seen in imatinib's Phase 3 IMPRES Study have not been observed at high incidence in TORREY Study 	1,4
M	Open-label Extension	 Patients continue to show meaningful improvement in PVR and 6MWD beyond 24 weeks of treatment, relative to treatment baseline, in ongoing OLE 56% (15/27) & 15% (4/27) of patients on drug for 72 weeks showed 20%+ & 50%+ improvement in PVR, respectively, versus 33% (9/27) & 0% of patients at week 24 	4

¹⁾ Frantz et al, Lancet Respiratory Medicine 2024.

demonstrated a +6.5m improvement in 6MWD relative to placebo, which did not achieve statistical significance. P-value for FC III patient population shown.

#TORREY Phase 2 Study was neither designed nor powered to achieve statistical significance in 6MWD. Seralutinib demonstrated a statistically significant improvement in 6MWD in Functional Class III (FC III) patients (n = 36), which was a pre-specified subgroup. In the overall population, seralutinib



²⁾ Frantz et al, World Symposium on Pulmonary Hypertension 2024.

³⁾ Zamanian et al, World Symposium on Pulmonary Hypertension 2024.

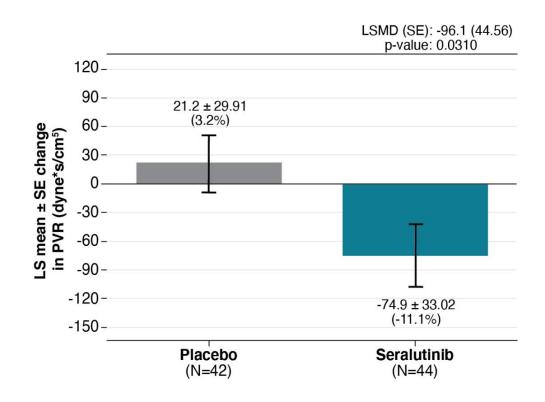
⁴⁾ McLaughlin et al, ERS Congress 2024. Patient 20%+, 50%+ PVR improvement data previously presented on investor OLE update call in December 2023.

PVR = pulmonary vascular resistance; RHC = right ventricle; PA = pulmonary arterial; mPAP = mean pulmonary arterial pressure; CT Scan = computed topography scan; OLE = open-label extension; wks = weeks; pbo = placebo.

*Statistical significance ("stat. sig.") defined as p ≤ 0.05. All improvements are relative to placebo. All p-values are week 24, unless otherwise noted. All p-values in this presentation are nominal, aside from primary endpoint (overall study population delta in PVR).

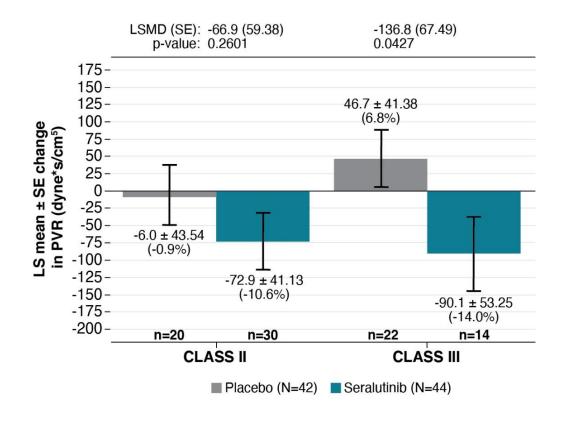
Primary Endpoint: Change in PVR From Baseline to Week 24

Overall Population



WHO Functional Class Breakout

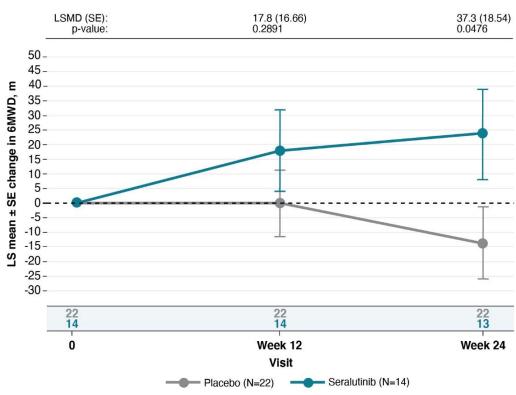
Pre-Specified Subgroup Analysis



Secondary Endpoint: Change in 6MWD from Baseline by Functional Class

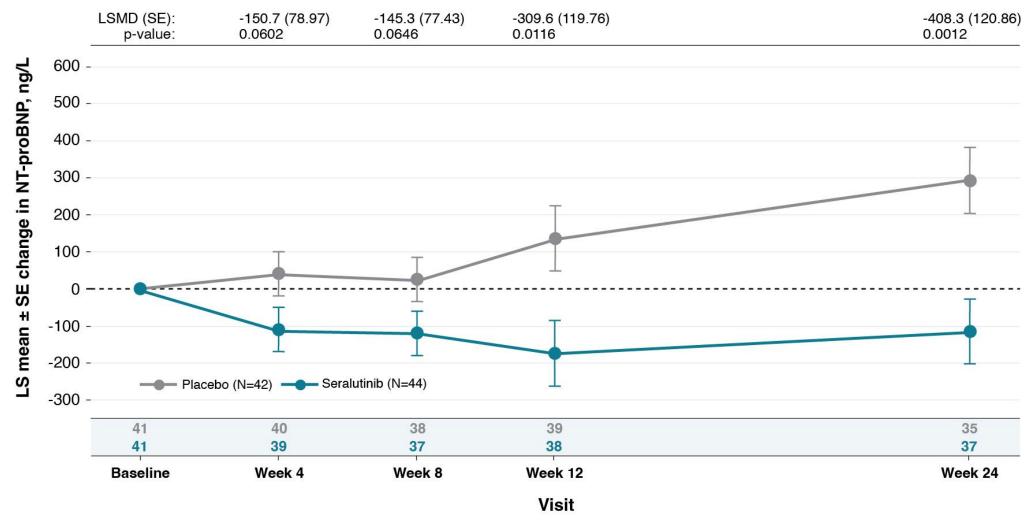
Overall Population LSMD (SE): 2.4 (10.87) 6.5 (12.26) p-value: 0.8273 0.5972 30 25 ± SE change in 6MWD, m 42 44 42 38 0 Week 12 Week 24 Visit Seralutinib (N=44)







Seralutinib Treatment Resulted in a Rapid and Sustained Reduction in NT-proBNP, a Biomarker of Right Heart Strain



Seralutinib treatment resulted in significant reduction in NT-proBNP vs placebo at Week 12 (-309.6 ng/L, p=0.0116) and Week 24 (-408.3 ng/L, p=0.0012).

Centrally-Read RHC and ECHO Results at Week 24 Consistently Favored Seralutinib (ANCOVA – Observed Cases)

Endpoint	LS Mean Difference (95% CI)	Statistically Significant Result Favoring Seralutinib (p ≤ 0.05)	Point Estimate Favoring Seralutinib	p-value
Right Atrium Area (cm²)	-1.99 (-3.783, -0.206)			0.0293*
RV Free Wall Strain (%)	-2.64 (-5.172, -0.098)			0.0420*
PA Compliance (mL/mmHg)	0.22 (0.009, 0.423)			0.0410*
RV Systolic Pressure (mmHg)	-8.10 (-13.877, -2.317)			0.0067*
PA Systolic Pressure (mmHg)	-6.98 (-12.774, -1.187)			0.0189*
PA Diastolic Pressure (mmHg)	-3.43 (-6.211, -0.643)			0.0165*
RV Fractional Area Change	2.62 (-1.405, 6.652)			0.1983
PVR index (dyne*s/cm ⁵ /m ²)	-160.42 (-333.970, 13.138)			0.0695
mRAP (mmHg)	-0.99 (-2.350, 0.367)			0.1503
Stroke Volume Index (mL/m²)	2.19 (-0.917, 5.299)			0.1644
Cardiac Index (L/min/m²)	0.13 (-0.100, 0.359)			0.2658



^{*} p ≤ 0.05

mRAP = mean right atrial pressure; PA = pulmonary artery; PVR = pulmonary vascular resistance; RV = right ventricle; LS = least squares; RHC = right heart catheterization; ECHO = echocardiography.

Frantz RP, et al. Lancet Respir Med 2024;12(7):523-534.

Incidence of TEAEs by Preferred Term: ≥ 5% in Seralutinib (Safety Population)

Preferred Term ^a	Placebo (N=42)	Seralutinib (N=44)
Number of subjects with a TEAE	36 (85.7)	41 (93.2)
Cough COVID-19	16 (38.1) 7 (16.7)	19 (43.2) 6 (13.6)
Diarrhea	3 (7.1)	6 (13.6)
Headache	8 (19.0)	6 (13.6)
Dizziness Fatigue	2 (4.8) 3 (7.1)	5 (11.4) 5 (11.4)
Nausea	6 (14.3)	5 (11.4)
Dyspnea Nightmare	5 (11.9) 1 (2.4)	4 (9.1) 4 (9.1)
Abdominal pain lower	0	3 (6.8)
Arthralgia	1 (2.4)	3 (6.8)
Back pain Chest discomfort	2 (4.8) 1 (2.4)	3 (6.8) 3 (6.8)
Nasal congestion	1 (2.4)	3 (6.8)
Nasopharyngitis	0	3 (6.8)
Rash	1 (2.4)	3 (6.8)
Throat irritation	0	3 (6.8)

All TEAEs in the table above were mild or moderate in severity.



TORREY Phase 2 Imaging Sub-Study: Assessing Pulmonary Vascular Remodeling in Patients Treated with Seralutinib

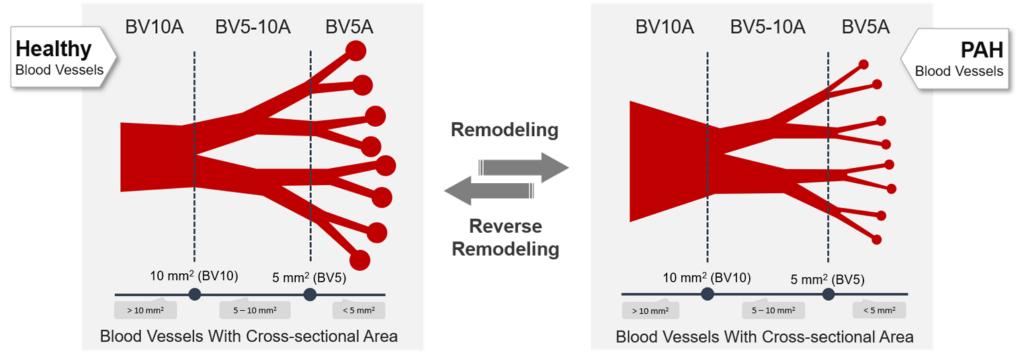
- Purpose: Provide evidence consistent with a reverse remodeling effect of seralutinib
- Hypothesis: volume of distal pulmonary arteries relative to volume of proximal pulmonary arteries will be increased by seralutinib as expressed by the ratio of BV5A to BV10A (BV510ARatio)
- Method: Thin-section, volumetric, non-contrast chest CTs were obtained, followed by automated pulmonary vascular segmentation



See publication - presented at ERS International Congress 2023 in Milan, Italy: "Seralutinib improves pulmonary arterial blood vessel volume distribution in pulmonary arterial hypertension (PAH): Results of the TORREY Phase 2 imaging substudy"



Pulmonary Vascular Volume of Small Distal Arterial Vessels is Decreased in PAH, Leading to Dilation of Larger Proximal Vessels



CT imaging can quantify these changes: BV5A: BVV of pulmonary arteries with a CSA < 5 mm²

BV5-10A: BVV of pulmonary arteries with a CSA between 5-10 mm²

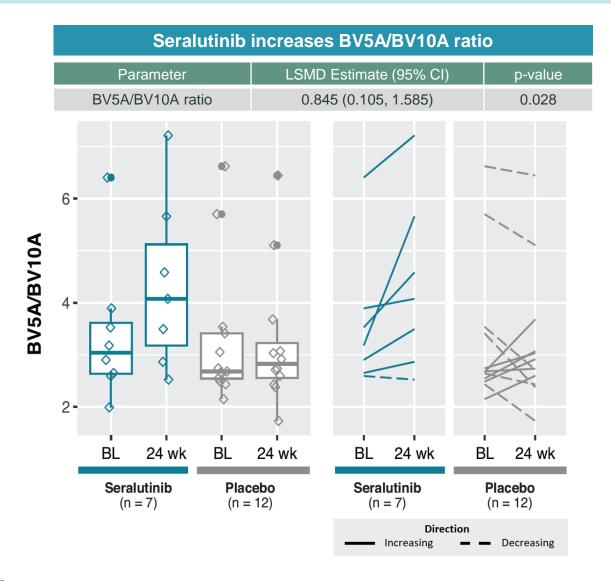
BV10A: BVV of pulmonary arteries with a CSA > 10 mm²

BV510ARatio: BV5A/BV10A

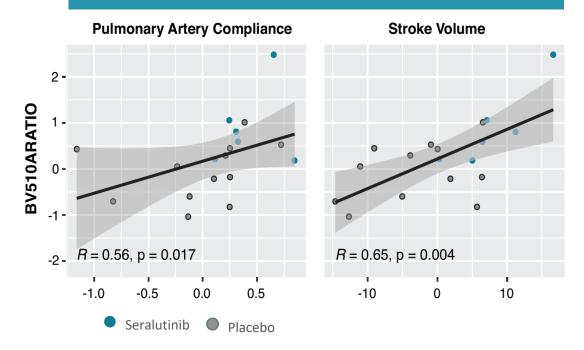
Pulmonary vascular pruning on CT correlates with histologic pulmonary vascular remodeling¹



Seralutinib Treatment Increases the BV5A/BV10A Ratio & Supports Blood Volume Redistribution Hypothesis

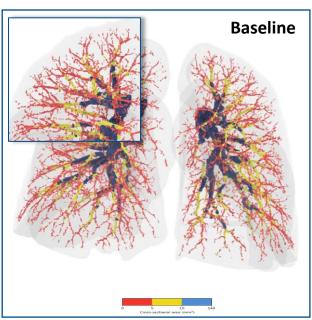


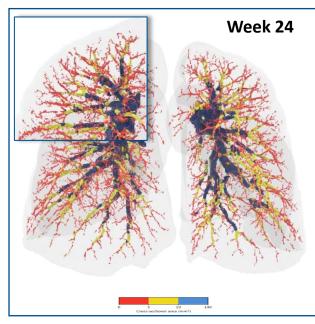
Change in BV5A/BV10A ratio from BL to Week 24 correlates with change in hemodynamics

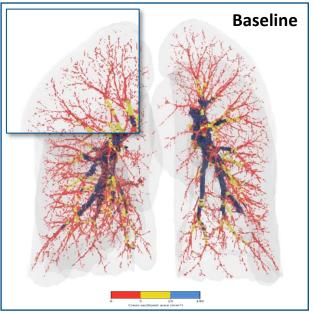


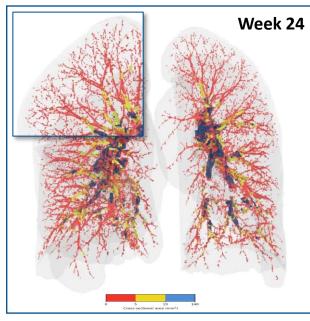


Examples of Imaging: Placebo vs. Seralutinib









Placebo patient

Female, 24 y, iPAH, FC II, treated with PDE5-i + prostacyclin

PVR change, dyne*s/cm⁵ (%) 283 (+65.4)

ΔBV510ARatio (% change) -0.70 (-28.9)

Seralutinib patient

Female, 58 y, iPAH, FC II, treated with ERA + PDE5-i + prostacyclin

PVR change, dyne*s/cm⁵ (%)

-159 (-39.0)

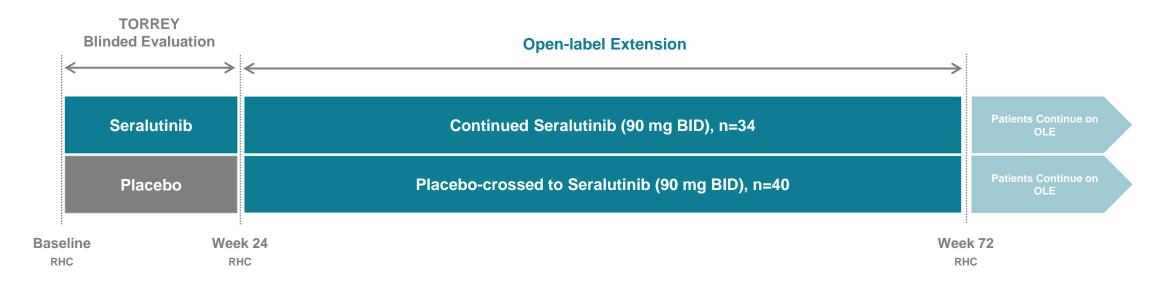
ΔBV510ARatio (% change)

+2.5 (+78.0)

The images shown are representative examples. The highlighted sections were chosen to illustrate changes in the pulmonary vasculature.



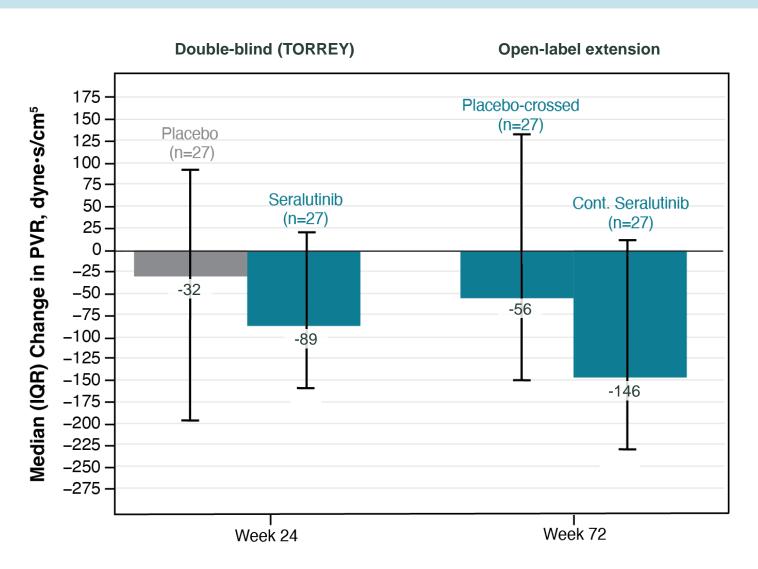
Ongoing TORREY Open-Label Extension



- Patient population: 73/80 patients who completed TORREY, 1 patient from a phase 1B study
- Objectives:
 - Ongoing, long-term safety & tolerability
 - Efficacy parameters, including hemodynamics at Week 72



PVR Continues to Improve With Seralutinib in the OLE

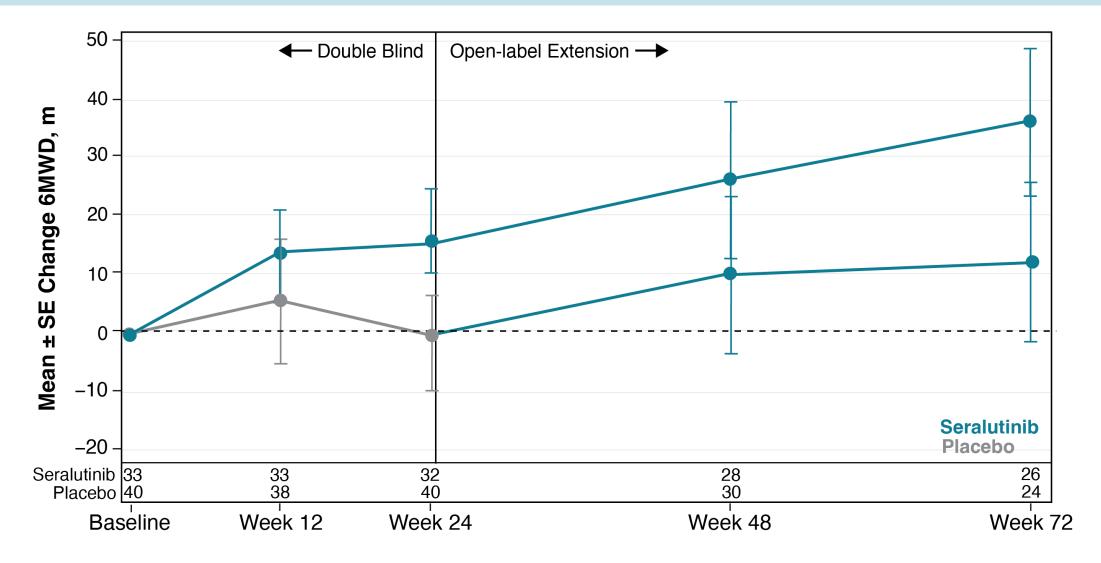


Median PVR Values, dyne*s/cm⁵

Visit	Placebo/ Placebo- Crossed	Seralutinib/ Cont Seralutinib
Baseline	650.0	620.0
Week 24	647.0	505.0
Week 72	603.0	475.0



6MWD Increases in Continued-Seralutinib Group & Placebo-Crossed Group





III. PROSERA Phase 3 Study

Ongoing PROSERA Phase 3 Study

Extended Double Blind Double Blind Placebo-Controlled Adults ≥ 18 and ≤ 75 years old **Treatment Period Treatment Period (24 weeks)** (up to an additional 24 weeks)* WHO Group 1 PH WHO Functional Class II or III Seralutinib 90mg BID + Background PAH Therapy PVR ≥ 400 dyne•s/cm⁵ N = 175Screening Baseline 6MWD 150 - 475m* (up to 4 weeks) Placebo BID + Background PAH Therapy or PVR ≥ 800 dyne•s/cm⁵* N = 175Week 24 Week 48 **Primary Endpoint** (6MWD) Evaluated at Week 24 **Open-Label Extension (OLE)** Subjects who complete Week 24 who experience a clinical worsening event may proceed directly to OLE



- Either REVEAL Lite 2 Risk Score ≥ 5 or NT-proBNP ≥ 300 ng/L
- Stable treatment with at least one SOC background therapy

PROSERA Key Inclusion Criteria



IV. The Next Frontier: PH-ILD

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

- WHO Group 3 PH is pulmonary hypertension associated with lung diseases and / or hypoxia
 - PH associated with interstitial lung disease (PH-ILD) is a subgroup of Group 3 PH
 - PH-ILD includes PH related to idiopathic pulmonary fibrosis (IPF)
 & PH related to connective tissue disease-associated interstitial lung disease (CTD-ILD)
- Characterized by pulmonary vascular pathology associated with PH, in addition to thickening & scarring of the lung interstitium resulting from ILD
- Only Tyvaso® is approved for PH-ILD, & only in the US
- Patients have poor disease prognosis & increased mortality rate as compared to PAH patients (40% 3-year survival rate²)









PH-ILD is an Ideal Next Indication for Seralutinib

Biologic Rationale:

Demonstrated Positive Impact on Reducing Pulmonary Hypertension

2

Clinical Trial Patient Dynamics are Favorable

3

High Unmet Need



- The pulmonary hypertension in PH-ILD is caused by the same proliferative, inflammatory, & fibrotic pathways as PAH
- Seralutinib demonstrated statistically significant improvement in PVR, right heart function/structure measures, & NT-proBNP in TORREY



- Lack of therapeutic options has fostered strong patient demand for clinical trials
- PH-ILD clinical trial patients have increased exercise impairment, as compared to PAH studies
 - Mean BL STELLAR (PAH) 6MWD: 401m
 - Mean BL INCREASE (PH-ILD) 6MWD: 260m
- Seralutinib demonstrated a stat. sig. pbo-controlled 38m increase in 6MWD in baseline FC III PAH patients* in TORREY (mean BL 6MWD = 367m)



- Only Tyvaso® is approved for PH-ILD, & only in the US and other select markets
 - No approved therapies in EU
- Patient population is potentially double the PAH population
- Patients have a high mortality rate, even compared to PAH

Phase 3 design to be discussed after interactions with global regulatory authorities

Seralutinb MoA Aligned with Underlying Pathophysiology of Group 3 PH

Disease Process	Cell Type / Mechanism	Potentially Relevant Pathway
Vascular Inflammation	Macrophages & ECs	CSF1RKIT
Vascular fibrosis	Fibroblasts / myofibroblasts	• PDGFR
Pulmonary vasculopathy (plexiform lesions)	Endothelial-to-mesenchymal transition	• PDGFR
Pulmonary arteriolar hypertrophy / hyperplasia	Pulmonary arteriole vascular smooth muscle cells	PDGFRBMPR2
Parenchymal interstitial lung	Fibroblasts	• PDGFR • CSF1R
inflammation & fibrosis	Epithelial-to-mesenchymal transition	• PDGFR
Shunt/hypoxia	V/Q mismatch	Multiple

Seralutinib Was Rationally Designed For PH & Is Highly Relevant For Targeted Indications



PH-ILD Presents a Significant Market Opportunity

	PAH	PH-ILD
US Prevalence	~30-50k ¹	~60-100k+ ³
Competitive intensity	16 marketed products	1 marketed product (US Only)
5-year survival rate	57% ²	23%4
Generics	8 generic products	0 generic products

Patients living with PH-ILD are deeply underserved



V. Milestones & Finances

Milestones & Financial Overview

Near Term Clinical Milestones

- Mid-2025: Commence Global Registrational Phase 3 Clinical Trial in PH-ILD
- 4Q 2025: Topline Data from Ongoing Phase 3 Clinical Trial in PAH, PROSERA Study

Financial Overview

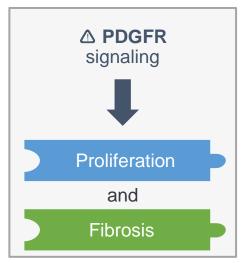
Cash, Cash Equivalents and Marketable Securities (As of 9/30/24)	~\$327mm
Principal of Convertible Notes Outstanding (As of 9/30/24; 5% annual interest; matures May 2027; conversion price: \$16.23)	~\$200mm
Common Shares Outstanding (As of 11/4/24)	~227mm

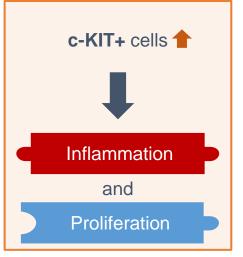


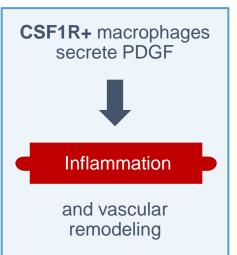
Appendix

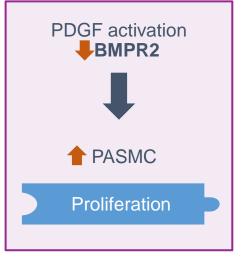
Contributing Factors to Vascular Remodeling

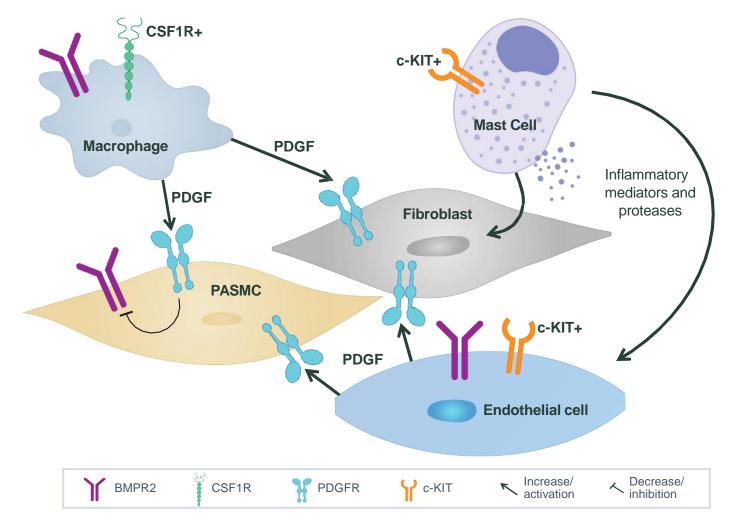
Role of PDGFR, CSF1R, c-KIT and Interactions with BMPR2













A Well-Suited Partner

 Global biopharmaceutical group with international R&D and commercialization infrastructure & operations, headquartered in Italy



- Over 85 years of experience, operations in >30 countries, >7,000 employees world-wide, including ~700 in R&D, and >€3 billion in revenue in 2023
- Chiesi's therapeutic focus perfectly aligns with seralutinib: AIR (respiratory disease), RARE (rare diseases), & CARE (specialty care, including cardiovascular disease)
- Global reach & areas of focus position Chiesi to enhance seralutinib's access to pulmonary hypertension (PH) patients across the globe



Encompasses products & services for the treatment of respiratory diseases among patients of all ages, from newborns to the elderly.

Asthma • COPD • PAH • IPF



Focusing on the treatment of patients living with rare or ultra-rare diseases.

Rare Immunologic Diseases



Combines products & services that support special care provided by medical professionals, as well as consumer healthcare/over the counter.

Cardiovascular Diseases



Value of Partnership to Gossamer

Provides Adequate Capital & Global Commercial Partner for Investment in Commercial Launch of PAH

- Bolstered Gossamer cash balance
- Gossamer & Chiesi can confidently invest in commercial planning during PROSERA study (expected Q4:25 topline readout)
- Chiesi is a global partner with significant commercial pulmonary & rare disease infrastructure

Accelerates Seralutinib into a Phase 3 Study in PH-ILD

- Pivotal Phase 3 Study in PH-ILD expected to begin in mid-2025, cutting years off potential development timeline
- Adds multi-billion-dollar peak sales opportunity in indication with high unmet medical need, strong biological rationale, & limited competition

Retained Strategic Optionality & Experienced, Motivated Partner

- Gossamer retains control over US commercialization & global development in PAH & PH-ILD
- Gossamer & Chiesi are committed to smart expansion into indications of unmet need that overlap with areas of expertise

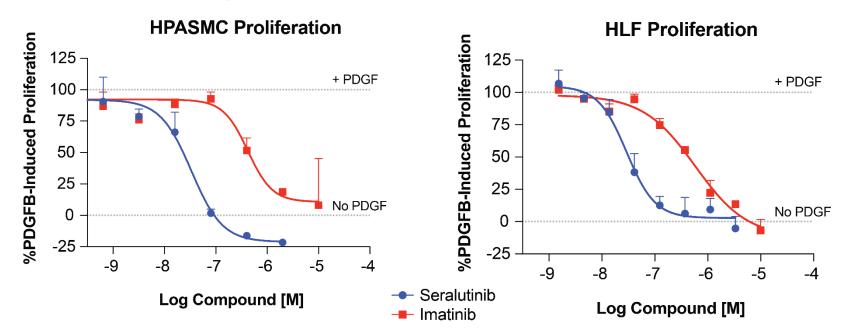


Seralutinib In Vitro Profile

Seralutinib is a potent PDGFR, CSF1R and c-KIT inhibitor

		Cell Ba	ased IC50 (nM)		
Compound	H1703 PDGFRα	HLF PDGFβ>α	PASMC PDGFRα=β	CSF1R	c-KIT
Seralutinib	32	29	33	8	8
Imatinib	62	579	419	1032	301

Seralutinib is highly potent in PASMC and HLF proliferation assays





In the Phase 3 IMPRES Study of Imatinib in PAH, Safety Liabilities Outweighed Clinically Meaningful Efficacy





Clinical Efficacy Results

Phase 3 IMPRES Study

- Primary Endpoint:
 - 6-Minute Walk Distance (6MWD)
 - 32-meter improvement (pbo-adj.)*
- Secondary Endpoint:
 - Pulmonary Vascular Resistance (PVR)
 - 32% reduction (pbo-adj.)*

Clinical Safety / Tolerability

Phase 3 IMPRES Study

- Adverse Events:
 - High rate of GI side effects
 - 44% SAE rate for imatinib group
 - 8 subdural hematomas across study and extension
- Discontinuations:
 - 33% for imatinib group, with most occurring in first 8 weeks of trial



Seralutinib Employs Multiple Strategies to Mitigate Imatinib's Liabilities

Molecule Specifically Designed for PAH

- Imatinib was developed & approved as an anti-cancer therapy
- Seralutinib utilized Phase 3 IMPRES learnings and targets underlying biology of PAH, including PDGFR α/β , CSF1R and c-Kit
- Seralutinib avoids c-ABL inhibition

Improved Selectivity Against Targets of Interest

- Increased potency* across target kinases v. imatinib
 - Increased potency against the PDGFRα isoform
 - Greater than ten-fold higher potency against PDGFRβ, c-Kit, and CSF1R

Designed for Inhalation

- Inhalation limits systemic exposure to mitigate systemic AEs, while directly getting drug to site of disease
- As part of inhalation process, some drug product is inevitably swallowed
 - Swallowed / ingested drug can enter systemic concentration
 - Seralutinib designed to have limited oral bioavailability (~5%)



TORREY Phase 2 - Baseline Demographics (ITT Population)

Characteristic	Placebo (N=42)	Seralutinib (N=44)	Total (N=86)
Age (years) – mean (SD)	49.5 (11.81)	48.3 (12.70)	48.8 (12.22)
Sex - n (%)			
Female	38 (90.5)	40 (90.9)	78 (90.7)
Male	4 (9.5)	4 (9.1)	8 (9.3)
Race - n (%)			
White	37 (88.1)	37 (84.1)	74 (86.0)
Black or African American	1 (2.4)	0	1 (1.2)
Asian	2 (4.8)	4 (9.1)	6 (7.0)
Other	2 (4.8)	3 (6.8)	5 (5.8)
Ethnicity – n (%)			
Hispanic or Latino	6 (14.3)	8 (18.2)	14 (16.3)
Not Hispanic or Latino	34 (81.0)	36 (81.8)	70 (81.4)
Not reported	2 (4.8)	0	2 (2.3)
Region – n (%)			
North America	30 (71.4)	29 (65.9)	59 (68.6)
Western Europe	10 (23.8)	11 (25.0)	21 (24.4)
Asia Pacific	1 (2.4)	4 (9.1)	5 (5.8)
Eastern Europe	1 (2.4)	0	1 (1.2)



TORREY Phase 2 - Baseline Background PAH Medication Use(ITT Population)

Characteristic	Placebo (N=42)	Seralutinib (N=44)	Total (N=86)
Number of background therapies – n (%)			
1	2 (4.8)	1 (2.3)	3 (3.5)
2	16 (38.1)	18 (40.9)	34 (39.5)
3	24 (57.1)	25 (56.8)	49 (57.0)
Prostacyclin/Prostacycin Receptor Agonist use			
- n (%)			
None	13 (31.0)	15 (34.1)	28 (32.6)
Monotherapy	1 (2.4)	1 (2.3)	2 (2.3)
Double therapy	4 (9.5)	3 (6.8)	7 (8.1)
Triple therapy	24 (57.1)	25 (56.8)	49 (57.0)
Parenteral Prostacyclin	19 (45.2)	19 (43.1)	38 (44.2)
Oral	10 (23.8)	10 (22.7)	20 (23.3)



TORREY Phase 2 - Baseline Disease Characteristics (ITT Population)

Characteristic	Placebo (N=42)	Seralutinib (N=44)	Total (N=86)
Age at PAH diagnosis (years) – mean (SD)	41.2 (11.65)	40.7 (15.84)	40.9 (13.87)
Years since PAH diagnosis – mean (SD)	8.78 (7.218)	8.07 (7.074)	8.41 (7.111)
PAH classification – n (%)			
Idiopathic	22 (52.4)	20 (45.5)	42 (48.8)
Heritable	5 (11.9)	10 (22.7)	15 (17.4)
Associated with:			
CTD	11 (26.2)	6 (13.6)	17 (19.8)
Anorexigen use	0	1 (2.3)	1 (1.2)
Methamphetamine use	4 (9.5)	4 (9.1)	8 (9.3)
Corrected congenital shunts	0	3 (6.8)	3 (3.5)
WHO FC – n (%)			
Class II	20 (47.6)	30 (68.2)	50 (58.1)
Class III	22 (52.4)	14 (31.8)	36 (41.9)
REVEAL 2.0 Risk Score ≥ 6 - n (%)	17 (40.5)	20 (45.5)	37 (43.0)
PVR (dyne*s/cm ⁵) – mean (SD)	661.3 (164.91)	675.8 (240.35)	668.7 (205.90)
6MWD (m) - mean (SD)	407.1 (107.02)	408.6 (75.11)	407.9 (91.54)
NT-proBNP (ng/L) – mean (SD)	645.6 (1158.75)	611.0 (714.58)	628.3 (956.83)



TORREY Phase 2 - Baseline Demographics and Disease Characteristics by Baseline WHO FC (ITT Population)

	Baseline WHO FC Class II		
Characteristic	Placebo (N=20)	Seralutinib (N=30)	Total (N=50)
Age (years) - mean (SD)	47.6 (11.69)	47.7 (13.42)	47.7 (12.63)
Female - n (%)	19 (95.0)	27 (90.0)	46 (92.0)
Race, White - n (%)	19 (95.0)	24 (80.0)	43 (86.0)
Region, North America – n (%)	13 (65.0)	20 (66.7)	33 (66.0)
Years since PAH diagnosis – mean (SD)	9.60 (7.262)	8.40 (6.961)	8.88 (7.034)
PAH classification – n (%)			
Idiopathic	11 (55.0)	16 (53.3)	27 (54.0)
Heritable	4 (20.0)	6 (20.0)	10 (20.0)
Associated with CTD	5 (25.0)	5 (16.7)	10 (20.0)
REVEAL 2.0 Risk Score ≥ 6 - n (%)	4 (20.0)	11 (36.7)	15 (30.0)
PVR (dyne*s/cm ⁵) – mean (SD)	638.3 (161.85)	689.9 (265.72)	669.3 (229.34)
6MWD (m) - mean (SD)	455.5 (63.96)	425.5 (62.98)	437.5 (64.45)
NT-proBNP (ng/L) – mean (SD)	406.8 (798.39)	609.9 (715.31)	525.3 (749.58)
On 3 background therapies – n (%)	11 (55.0)	18 (60.0)	29 (58.0)
ERA + PDE-5i + Prostacyclins/PRA	8 (40.0)	16 (53.3)	24 (48.0)
ERA + sGC + Prostacyclins/PRA	3 (15.0)	2 (6.7)	5 (10.0)

Baseline WHO FC Class III		
Placebo (N=22)	Seralutinib (N=14)	Total (N=36)
51.1 (11.94)	49.4 (11.40)	50.4 (11.60)
19 (86.4)	13 (92.9)	32 (88.9)
18 (81.8)	13 (92.9)	31 (86.1)
17 (77.3)	9 (64.3)	26 (72.2)
8.02 (7.263)	7.36 (7.527)	7.76 (7.266)
11 (50.0)	4 (28.6)	15 (41.7)
1 (4.5)	4 (28.6)	5 (13.9)
6 (27.3)	1 (7.1)	7 (19.4)
13 (59.1)	9 (64.3)	22 (61.1)
682.2 (168.62)	645.7 (179.29)	668.0 (171.25)
363.2 (120.05)	372.4 (87.97)	366.8 (107.43)
873.0 (1403.06)	613.3 (742.17)	773.7 (1187.34)
13 (59.1)	7 (50.0)	20 (55.6)
10 (45.5)	6 (42.9)	16 (44.4)
3 (13.6)	1 (7.1)	4 (11.1)